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FLUID VOLUME CONTROL IN HEART-LUNG BYPASS OPERATIONS IN OPEN-HEART SURGERY

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In this article 25 consecutive heart-lung bypass operations are discussed; these are cases 7 - 31 of those performed in Johannesburg. Only 13 of them are considered in the post-operative studies discussed, because in 6 cases the records were incomplete on account of bed-wetting and spillage, and 6 patients died.

Before the chest is opened, a polythene tube is inserted into a vein at the elbow and advanced into the superior vena cava, and a similar tube passed into the inferior vena cava from a vein in the groin. The former is used for the measurement of venous blood pressures and the latter for the administration of blood and fluid. A polythene tube inserted into the brachial artery is used for monitoring the arterial pressure.

The venous blood pressure is measured in the superior vena cava to preclude the build-up of a high venous pressure in the head, with its attendant dangers. The tube is attached to a 3-way tap, into which leads a drip of 5% dextrose in water. Into the remaining outlet of the tap a 2-foot piece of blood-transfusion tubing is fitted. This is attached vertically to a drip-stand, in front of a centimetre scale marked on adhesive tape, of which the zero mark, determined with a spirit level, is at the level of the right auricle. Continuous readings of the venous pressure throughout operation are thus possible. The tubing thus arranged is used post-operatively in the ward; by placing the 3-way tap at the side of the chest on a level with the right auricle, and measuring the column of water above it with an ordinary ruler, the venous pressure can be read whatever the position of the patient. The femoral venous catheter is also left in for as long as it is required for the administration of blood.

On rare occasions, while the chest is pulled open by retractors, the veins in association with the superior vena cava become stretched or kinked. Under these circumstances the fluid level in the venous manometer becomes raised, fixed and inaccurate. We have found that the venous-pressure readings in the inferior and the superior vena cava are the same so that, where difficulty is encountered in obtaining satisfactory readings in the superior vena cava, the inferior vena cava is used. Whichever tube is not being used for measuring the venous pressure is used for the administration of blood.

BLOOD VOLUME CONTROL

During the Operation

Blood loss during the operation is estimated from the following sources:

1. Blood is sucked from the operation site into two 2-litre measuring cylinders under the operating table.

2. Swabs are weighed before autoclaving, and the weight is written on the swabs. After use, the swabs are again weighed, and the original weight is subtracted from the weight of the blood-stained swabs to ascertain the weight of blood lost.

3. A check is kept throughout on the volume of blood removed for the purpose of laboratory tests.

4. The quantity of blood lost in spillage is ascertained by wiping up the blood lost with a swab of known weight and weighing the blood-soaked swab.

5. Towels used at the operation site are weighed before and after operation.

6. The amount of saline used during the operation for moistening the heart is subtracted in arriving at the total quantity of blood and fluid lost.

The policy has been to keep the volume of blood transfused ahead of the volume lost. An extra amount of blood is given during the operation to replace the blood lost to the circulation, which cannot be immediately assessed. This loss, which occurs at the operation site, into the pericardial and pleural cavities, and in redistribution within the body, is recognized by the fall in venous and arterial pressures. The volume of blood needed is that required to keep the blood pressure at about the pre-operative level. In children of up to 50 lb. in weight a lead of up to 100 ml. of tranfused blood is maintained over and above the blood lost. In children of 50 - 100 lb. in weight a lead of 100 - 200 ml. of blood is maintained. In patients of over 100 lb. in weight the usual lead required is 200 - 400 ml. On 3 occasions the blood pressure was maintained to the end of the operation with a transfused blood volume which was less than the volume of blood lost; these patients had marked peripheral vasoconstriction.

The total quantity of blood lost during operation is represented in Fig. 1, which shows an average loss of 1,265 ml. The greatest quantity lost was 2,680 ml., and the smallest quantity was 500 ml. The average volume of blood lost into the suction pump was 898 ml., and that removed by the pathologists, 142 ml.; the average weight of blood lost in swabs was 152·4 g.; and that spilt was 74 gm. The average volume of blood replaced during the operation was 1,469 ml., showing an average positive balance of 204 ml. The average volume of heparinized blood used in replacement was 548 ml., and of citrated blood 921 ml.

Intravenous citrated blood is used to replace the blood lost before and after bypass and in the post-operative period. During the bypass heparinized blood poured directly into the oxygenator reservoir is used for replacement. Measurement of the arterial and venous blood pressures are much more informative in maintaining the correct blood volume during bypass and after bypass than a knowledge of the exact balance of blood loss and replacement. In the absence of any marked haemorrhage a flow rate of 2·2 litre per minute per square metre (sq.m.) of body surface area (BSA),[1] a mean

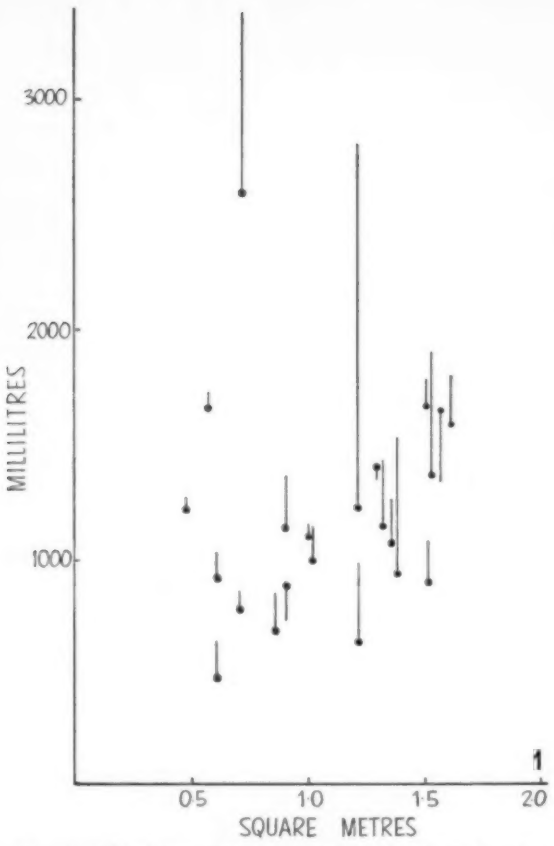

Fig. 1. Blood loss and replacement during operation, by sq.m. BSA. The dot denotes the volume of blood lost and the point at the end of the vertical line the volume replaced.

arterial blood pressure of 70 mm. Hg, and a venous blood pressure of 7 - 10 cm. H_2O, will produce a haemodynamic state in which the blood volume will be automatically regulated.[2] It is nevertheless valuable to keep the blood balance accurately throughout the operation for, in the event of a large haemorrhage, the exact amount of blood lost is known as it is lost, and is replaced immediately, so that the normal haemodynamics are preserved and shock or over-perfusion prevented. Rapidly fluctuating blood-pressure levels are of serious consequence in that they cause peripheral vasoconstriction at the end of bypass. If the bloodpressure falls during or after bypass, it should be raised to the desired level as smoothly and speedily as possible and, if it rises to too high a level, it should be as gently and speedily lowered. A high blood pressure during bypass is indicative of an over-perfusion state and too great a volume of blood in the patient, with its dangers of cerebral haemorrhage and pulmonary infarction. A high blood pressure after bypass is indicative of too big a circulating blood volume, with its attendant danger of heart failure.

During bypass, with the flow rate kept at 2·2 l. per sq. m. BSA per minute, the arterial blood pressure and venous blood pressures are kept at their respective desired levels by raising or lowering the venous drainage reservoir. Under this set of circumstances the circulating blood volume in the patient will remain normal and constant. If the blood level in the oxygenator falls, it should be topped up by the addition of heparinized blood to the oxygenator. The blood thus added to the pump oxygenator to maintain these pressures ensures the replacement of blood lost from surgery and the blood which often seems to transfer from the oxygenator to the patient as bypass proceeds beyond 30 minutes.[2] The latter represents a volume which is transferred to the patient and which cannot be accounted for purely on the basis of blood loss.

It is thus seen that, apart from the addition of blood to the pump oxygenator, regulation of the height of the venous drainage reservoir plays an essential part in maintaining the correct blood volume in the patient. During bypass, should the arterial blood pressure fall, more blood is needed in the patient to re-establish the pressure; this is achieved through cutting down the outflow of blood from the patient by raising the venous drainage reservoir. The blood that is being supplied by the pump is kept at the constant optimum flow, and in this way the amount of blood in the patient is increased, with a consequent rise in both the arterial and venous blood pressures. Should the blood pressure become unduly elevated, the venous reservoir is lowered, giving a greater drain-off of blood; thus, if the flow rate is kept constant, the volume of blood in the patient is diminished and the arterial and venous blood pressures are reduced to the desired levels. A low arterial blood pressure, low venous pressure, high flow rate, and high arteriovenous oxygen extraction, denote an inadequate volume of blood in the whole pump-oxygenator-patient system, which is only correctable by the addition of extra blood to the oxygenator. A high arterial blood pressure, high venous pressure, and low flow rate, denote too great a volume of blood in the system. This is corrected by lowering the venous reservoir and allowing the oxygenator blood level to rise.

Water Administration during Operation

As little as 100 - 150 ml. of 0·9% sodium chloride or 5% dextrose in water was administered during the operation. This was kept low intentionally, for it was felt that earlier patients had developed a raised jugulovenous pressure from having been given too much fluid. This restriction is now felt to have been unnecessarily severe, and up to 1,500 ml. of 5% dextrose in water, depending on the size of the patient, will henceforth be administered during the operation. The insensible loss of water during chest operations has been stated to be of the order of 2·3% of the body weight in 2 hours of operation.[3]

Post-operative Blood Requirements

During bypass, on occasions, considerable peripheral vasoconstriction occurs. In some of these cases the blood pressure is being maintained by a blood volume which is below that of the patient's total blood volume before operation. Such patients require the gentle addition of more blood to stimulate the reversal of the shock state and, as the peripheral arterial and venous tree dilates to normal, the additional blood is available to maintain the circulating blood volume. Another shock state which may develop is that of peripheral vasoconstriction with falling blood pressure due to a redistribution of blood, with pooling possibly in

the viscera. This may necessitate the administration of blood well in excess of the estimated loss, in order to maintain the patient's circulating blood volume and blood pressure. As this state of shock wears off, the body overcomes this central pooling effect, and more blood is returned to active circulation. This results in cardiac overload, with left ventricular failure manifested by cough, wheezing, rhonchi and crepitations, which occur on the 2nd to the 4th post-operative day. A mercurial diuretic is most effective in combating this. This late overload is also a possible explanation of the raised jugulovenous pressure seen some days after operation, which is best treated with digitalis and diuretics.

Another peculiar state, which becomes less frequent as experience in heart-lung work increases, develops in the first 48 hours. The clinical picture comprises pallor, cold extremities, peripheral vasconstriction, and high rectal temperature rapidly progressing to hyperpyrexia. To me, the best explanation of this state is a redistribution of blood, with pooling in the viscera and depletion of the circulating blood volume. The response of the sweat glands becomes progressively less as the rectal temperature rises, and a critical point can be reached at which sweating stops and cooling ceases, with rapid and, without treatment, fatal results.[4] The treatment is the administration of sufficient blood to maintain the blood pressure and the institution of measures to curtail the hyperpyrexia, which should include a 4 oz. enema of ice water, cold sponging, and a cool room with the minimum of clothing. Incompatibility of blood is offered as an explanation of this syndrome. This may be right, but the peripheral arterial and venous constriction must be due to an associated diminution of the circulating blood volume, because hyperpyrexia also occurs in patients who are flushed and warm to the touch. This hyperpyrexia responds well to steroids.

The maintenance of a record of the loss and replacement of blood is essential in the post-operative period, for the following reasons:

1. To act as a check that the correct blood volume is being maintained.

2. If it becomes apparent that large volumes of blood are required to maintain the blood pressure, the presence of an internal haemorrhage must be suspected and sought after.

3. It is as well to have a record of the extra blood administered so that, when pooling passes off and blood is made available for circulation, the cardiac failure which may ensue can be treated with a fuller understanding.

A low venous-pressure reading is always an accurate reflection of the right auricular pressure, and this, together with a low arterial pressure, is an indication for administering more blood. A high venous pressure, on the other hand, if associated with a low arterial pressure, may denote heart failure, or cardiac tamponade from bleeding into the pericardial cavity. But in shock associated with intense constriction of the veins, the opening of the intravenous tube used for measuring the venous blood pressure may be gripped tightly by the vein, resulting in a false high reading, which may mislead one into withholding blood when it is an urgent necessity.

Size of Patient

We have never weighed our patients before and after operation, for lack of a suitable scale. A comparison of the pre-operative and post-operative weights would be helpful. A post-operative weight less than the pre-operative weight would denote a definite deficit of blood or water; but a post-operative weight equalling or exceeding the pre-operative weight would give no indication of the adequacy of the

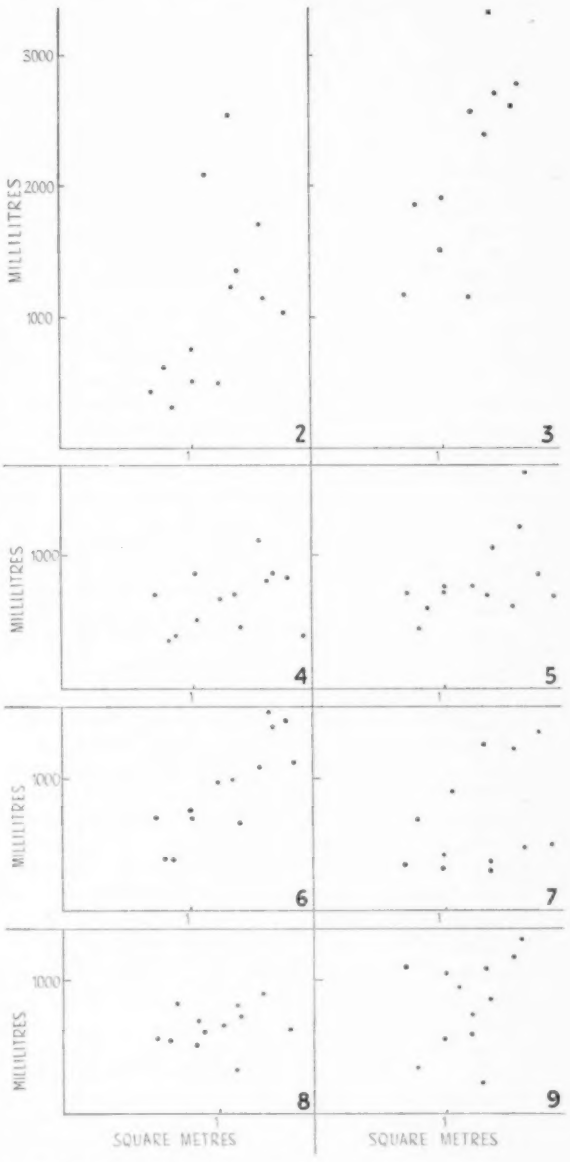

Fig. 2. Post-operative blood loss.
Fig. 3. Blood loss during and after operation.
Fig. 4. Fluid intake in the first post-operative 24 hours.
Fig. 5. Fluid intake in the second post-operative 24 hours.
Fig. 6. Fluid intake in the third post-operative 24 hours.
Fig. 7. Urine output in the first post-operative 24 hours.
Fig. 8. Urine output in the second post-operative 24 hours.
Fig. 9. Urine output in the third post-operative 24 hours.

circulating volume, the extent of redistribution, or the amount of blood lying free in the chest from haemorrhage, which can only be satisfactorily estimated by frequent X-ray examinations.

The size of the patient bears no relationship to the amount of blood lost during an operation. Fig. 1 shows that as much blood may be lost during the operation on a small child as on an adult. On the other hand, in the post-operative period, the amount of blood lost is roughly proportional to the size of the patient. The bigger the patient, the greater the anticipated blood loss. In a patient of 1 sq.m. BSA the anticipated post-operative blood loss is in the neighbourhood of 500 ml. In a patient of 1·5 sq.m. BSA the anticipated post-operative blood loss is of the order of 1,200 ml. (Fig. 2). Nearly all the post-operative blood drainage from the chest takes place in the first 12 hours. The total blood loss during and after the operation increases with the size of the patient (Fig. 3); the difference is attributed to the greater loss in the bigger patients after operation.

Post-operative Fluid Intake

Fluid in the post-operative phase was administered as 5% dextrose in water, intravenously, and as water by the mouth when the patient was able to swallow. The water intake in the first 24 hours (Fig. 4) averaged 524 ml. per sq.m. BSA. In the second 24 hours the average was 690 ml. per sq.m. BSA—a slight increase over the first 24 hours (Fig. 5). The amount of water administered in the third 24 hours was greater in the bigger than in the smaller patients (Fig. 6), the average being 727 ml. per sq.m. BSA. It is felt that these patients have been kept unnecessarily dry, and up to 1,500 ml. of 5% dextrose in water is to be administered to the patient in the early stages of operation and a 50% increase in the daily intake of fluid is planned. The following are comparative figures: (1) Sturtz *et al.*[5] administered 500 ml. per sq.m. BSA on the first post-operative day, 750 ml. on the second day, and 750 ml. on the third day. The patients were nursed in an oxygen tent with the humidity kept as near to 100% as possible, which reduced the insensible loss to a minimum. (2) Cleland[6] administered 400 ml. per sq.m. BSA on the first post-operative day and 800 ml. on the second day. Thereafter the patients were allowed to take fluids by the mouth as they wished. (3) Phillips *et al.*[7] administered 1·5 - 2 ml. per kg. of body weight per hour. (4) Heeley, *et al.*[8] in a survey of children with different illnesses who were in bed in the wards at a temperature of 76°F, calculated that the average insensible loss of water per sq.m. BSA per day was 1,200 ml. in children 0 - 3 years old, 950 ml. in those 3 - 8 years old, and 700 ml. in those of 8 years and older.

Urine Output

The patients passed urine before the operation and were not catheterized after the operation. There is no absolute relationship between the size of the patient and the output of urine in the first 24 hours (including that passed during the operation); the bigger patients who secreted more urine (Fig. 7) had bigger intakes during the operation, because greater liberty was taken with a possible overload of fluid in the bigger patients than in the smaller. The average quantity of urine secreted in the first post-operative 24 hours was 505 ml. per sq.m. BSA. In the second 24 hours, the volume of urine (Fig. 8) secreted was more uniform for all patients; the average volume was 562 ml. per sq.m. BSA. In the third 24 hours (Fig. 9) there was a tendency towards a greater output in the bigger patients; the average output was 505 ml. per sq.m. BSA.

Proteinuria, Glycosuria, Urine Specific Gravity

Protein in the urine was found post-operatively in 3 patients (out of 13), +++ in one and + in the other two. The protein disappeared in all 3. Sugar was not detected in the urine on any occasion.

The specific gravity of the urine was, on the whole, high. During the first 3 post-operative days, in 6 cases it was 1,030 or over, in 4 cases 1,025 – 1,029, and in 3 cases 1,020 – 1,024. The highest values were seen on the first post-operative day, but subsequent values were also high. This denotes that these patients were being kept dry, which, indeed, was the intention. There does not seem to be good reason for keeping the patients quite so dry, and henceforth they are to be given more fluid.

SUMMARY

The control of blood and fluid volume in patients undergoing open heart surgery, both while on the heart-lung oxygenator and during the post-operative 3 days, is considered.

The blood and fluid volumes in 25 consecutive cases are analysed.

I wish to thank the thoracic surgeons (Mr. D. I. Adler, Mr. L. Fatti, Mr. D. N. Fuller and Mr. P. E. Marchand) for allowing access to their patients; and also the physicians (Dr. J. L. Braudo and Dr. M. M. Zion), the anaesthetists (Dr. F. J. Durham, Dr. C. Frost, Dr. K. B. Meaker and Dr. C. H. Van Hasselt), and Dr. H. B. W. Greig and Mr. L. A. du Plessis (thoracic surgeon), for their help.

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FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

The Fifth International Poliomyelitis Conference, organized by the International Poliomyelitis Congress and sponsored by The National Foundation, USA, and the Danish National Association for Infantile Paralysis, will be held in Copenhagen on 26 - 28 July 1960.

The Conference scientific sessions will be divided into two parts, on virology and on poliovirus vaccines. The presentation of papers has been arranged by invitation, but time will be allowed for general discussion of these papers. There will also be scientific and technical exhibits.

The proceedings of all sessions, including all scientific papers presented, and a summary of the discussions and a short description of the scientific exhibits will be edited and published as the official proceedings of the Conference. The official languages of the Conference will be English, French, German, Russian and Spanish.

Registration and reservation of accommodation should be made before 1 April if possible. Correspondence and inquiries should be addressed to the Secretariat, 5 Tuborgvej, Hellerup, Denmark. Telegraphic address 'Poliocon'. A limited number of brochures and reservation forms are available from the office of the *Journal*, brochures are also available from the Manager of the Scandinavian Airlines System, P.O. Box 1706, Cape Town (501 Trust House, Foreshore).

South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

ANTICOAGULANT THERAPY

The treatment of thrombotic and potentially thrombotic states with anticoagulant drugs has now become commonplace. Numerous patients receive this form of therapy for acute conditions, and the number of patients who are being maintained on continuous long-term therapy for chronic cardiovascular disease is growing daily.

The technical problems of anticoagulant therapy are relatively simple. For rapid treatment of an acute condition intravenous heparin is unsurpassed, as is deep subcutaneous or intramuscular concentrated heparin during the following 48 hours. There is, as yet, no oral form of heparin which has therapeutic value. The administration of oral anticoagulant drugs is started simultaneously with the first dose of heparin and forms the basis of maintenance therapy. There appears to be little to choose between the various oral preparations in common use. The original dicoumarol is still widely used, but in South Africa and Britain phenindione (dindevan, indema, hedulin) is perhaps the most widely used drug of this group. Warfarin sodium (coumadyn, marevan) has gained in popularity in recent years, and there are other satisfactory drugs as well. The main point would appear to be to learn to know one of these drugs well, much as one gets to know one or two drugs of the digitalis or antihistamine groups. Many of the drugs in common use are satisfactory if used intelligently.[1]

The dose should not be varied excessively since too frequent tests may be as dangerous as too few. There is a tendency to attach too much importance to the actual figures of the prothrombin estimation. It matters little if the estimation is expressed as a percentage or an index; what is important is to know the therapeutic range, and this is sometimes quite wide. If anything, one should perhaps err on the side of keeping the prothrombin too high rather than too low, though there is evidence that too lax a control can also be dangerous.[2] The accepted practice is to keep the figure as low as can be done with safety, but a much higher level might possibly be equally satisfactory. A fixed dose is the ideal, and many patients have received the same dose for years. Once the readings are reasonably stable the dose need not be varied by more than perhaps half to one tablet per week. It is often better to check a divergent reading than to vary the dose. If the prothrombin reading is near the therapeutic levels the dose should not be altered at all. It is only when repeated readings are well outside optimum levels that the dosage schedule need be revised. Once the patient is well stabilized, tests need be done only once every two to three months.

An important cause of a divergent reading is that the patient has omitted to take the prescribed dose. It is not easy to remember to take a pill every day and, sooner or later, even the most conscientious pill-swallower begins to doubt whether he did in fact take his medicine. A useful trick is to advise the patient to put a week's supply of pills in one bottle and to take these pills during the week. The dose on the seventh day is whatever remains in the bottle!

There are surprisingly few contraindications to anticoagulant treatment. Severe bleeding diathesis, liver disease, ulceration in the gastro-intestinal tract, and possibly pregnancy are perhaps the most important contraindications. It is useful to remember that unexpected haemorrhage, when the prothrombin is not too low, is an indication for search for a local cause of bleeding.

It is probably wise to interrupt treatment if surgery is required. Major surgical operations without excessive blood loss, performed while the patients are being treated with such drugs as dicoumarol,[3] have been reported, but in general this is not advised. There is a difference, too, between patients who have been on these drugs for only a week or two and those who have received treatment for an extended period. Other coagulation factors, like Christmas factor, may become depressed and this will not necessarily be reflected in the prothrombin reading. Minor surgery, e.g. dental extraction, can usually be performed with safety two or three days after stopping phenindione, and the administration of the drug can be restarted within twenty-four hours after the operation when most of the pills which had been omitted will probably still be required. For emergency use vitamin K_1 is available, but it should only rarely be needed.

Are all the efforts and cost which are currently being expended in the therapeutic application of these drugs worth while? It would appear that this type of treatment has some value, though the proof is far from being satisfactory despite the many years which have elapsed since its introduction into medicine. It would also appear that a patient, who is admitted to hospital for an acute coronary thrombosis, has a better chance of leaving the hospital alive if he is given anticoagulant therapy than if this is not done. This might be due to the prevention of pulmonary embolism rather than the prevention of further coronary thrombosis. If this is the case the treatment would appear to be equally justifiable for any patient with chronic congestive cardiac failure or even for an (elderly) person who has to remain in bed. In chronic coronary artery disease there is room for even more scepticism despite some enthusiastic reports.[4-6] A recent MRC trial[7] has found this treatment beneficial, but some authors remain unconvinced.[8]

Anticoagulant treatment does appear to be of value in the prevention of cerebral embolism in chronic rheumatic heart disease,[9] but its use in the immediate treatment of cerebral embolism or in patients with cerebral thrombosis is not clearly established, although some reports tend to suggest that it might be of help.[10-12] It is not clear how long this treatment of patients with established vascular disease should continue—for the rest of their lives, or for a year or perhaps less?

These questions require an urgent answer and a clear case would appear to exist for a properly controlled study along these lines. The number of patients for whom this treatment is being prescribed is growing at an alarming

rate. Let us hope that clear-cut answers will be obtained before we are overwhelmed by the avalanche which is now rapidly gaining speed.

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KODE VIR GEBRUIKE VAN VERKOOPSBEVORDERING

Gedurende die afgelope paar dekades het die hele grondslag waarop die mediese praktyk gebaseer is, in ons eie land sowel as oor die res van die wêreld, ingrypende veranderinge ondergaan. Hierdie veranderinge spruit uit ontwikkelinge op die gebied van die mediese wetenskap self, sowel as uit die feit dat dit vir die farmaseutiese firmas moontlik geword het om kragtige en doeltreffende geneesmiddels op ongeëwenaarde skaal te vervaardig.

Een van die belangrike praktiese probleme wat uit hierdie toestand van sake ontstaan het, is die vraag: Hoe kan die informasie wat die groot aantal farmaseutiese firmas oor hul produkte beskikbaar het, aan die dokter oorgedra word sodat hy in staat is om dit op die hoogste vlak en in die beswil van sy pasiënte te gebruik? Die verskillende firmas het hul eie antwoorde op hierdie vraag gevind of probeer vind. Hulle het naamlik tegnieke van verkoopsbevordering ontwikkel wat die volgende insluit: Die verspreiding van informasie deur verteenwoordigers wat die dokter in sy huis en by sy spreekkamer en by die hospitaal besoek, direkte versending van advertensies en geskrewe informasie aan dokters, tydskrifadvertensies, rolprente, ens.

In die verlede is daar dikwels bedenkinge uitgespreek deur die dokters sowel as deur die verantwoordelike hoofde van die firmas self oor hoe betroubaar en onfeilbaar die verskillende tegnieke van verkoopsbevordering is. Die Ethical Drug Association of South Africa, waarvan een-en-veertig farmaseutiese firmas lede is, het nou 'n besondere prysenswaardige stap gedoen deur 'n ‚Kode vir verkoopsbevorderingsgebruike vir etiese geneesmiddels' op te stel waarin hulle self die beginsels uiteensit op grond waarvan hulle reken dat optrede in die verkoopsveld op die hoogs moontlike ‚etiese' vlak kan geskied. Die kode is in die twee landstale gedruk en word ter inligting van lede van die Mediese Vereniging in die vorm van 'n spesiale brosjure in hierdie uitgawe van die *Tydskrif* ingevoeg.

Namens die mediese professie wil ons die Ethical Drug Association of South Africa gelukwens met hierdie progressiewe stap en die hoop uitspreek dat dit sal bydra tot 'n volkome bevredigende verstandhouding tussen lede van die mediese professie en lede van die Ethical Drug Association; ook dat dit uiteindelik sal bydra tot die ontwikkeling van diens aan pasiënte op die hoogste vlak.

CODE OF SALES PROMOTION PRACTICE

During the past few decades the basic pattern of medical practice, in our own country and in the rest of the world, has undergone radical changes. These changes resulted from the rapid developments in the medical sciences and from the fact that pharmaceutical firms have been able to produce effective and potent drugs on an unparalleled scale.

One of the important practical problems which has arisen from this situation is the question: In what manner can the pharmaceutical firms best convey the available information about their products to doctors so as to enable them to use the information to the best advantage of their patients? Different firms have attempted to find their own answer to this question. They have, for instance, developed a number of techniques for their promotional activities, such as the dissemination of information by representatives, who visit the doctor at his house, at his consulting rooms, or at the hospital; dispatching advertisements by direct mailing; journal advertising; films, etc.

Doubt has often been expressed in the past by doctors and by responsible heads of firms in regard to the reliability and infallibility of these techniques of sales promotion. In an attempt to approach this problem in a constructive way, the Ethical Drug Association of South Africa, which consists of forty-one member firms, has now taken a most commendable step in formulating a 'Code of sales promotion practice for ethical medical products' in which the principles are defined according to which promotional activities could be carried out on the highest possible ethical level. A brochure containing the code has been printed in both official languages and is inserted in this issue of the *Journal* for the information of members of the Medical Association.

On behalf of the medical profession we wish to congratulate the Ethical Drug Association of South Africa on this progressive step and we wish to express the hope that the adoption of this code will lead to a completely satisfactory relationship between the members of the medical profession and members of the Drug Association. We hope, too, that it will eventually contribute towards the development of the highest possible standard of service to patients.

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PHAEOCHROMOCYTOMA IN A CHILD: A CASE WITH SOME UNUSUAL FEATURES

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The diagnosis of phaeochromocytoma in children is becoming increasingly frequent and 42 cases have been reported in children under the age of 14 years. Daeschner *et al.*,[1] in 1954, reviewed 17 cases, and 2 years later a review of 24 cases by Moore and Shumacker[2] appeared and 2 further cases with successful operative removal were reported by Robinson and Williams.[3]

A number of comprehensive reviews of phaeochromocytoma in childhood have appeared in the literature.[4,2,5] Of the 24 cases in Moore and Shumacker's list,[2] 6 are not recorded among the 32 reported by Smid and Du Shane.[4] To these are to be added the case reported by Higgins,[6] and 3 familial cases reported by Cone *et al.*[5] One of the last-mentioned 3 patients, a boy, required a further operation 29 months after the first, for the removal of a second tumour occurring in the neck.[7] Popper and Theron,[8] in 1954, reported a case of a phaeochromocytoma in a Bantu child in Johannesburg. It appears that the case presented here is thus the second one under the age of 14 years described in South Africa.

In dealing with a case of hypertension, any medical student will include a phaeochromocytoma in his differential diagnosis, though the incidence of this tumour is very small. In the majority of reports on the condition in childhood, persistent hypertension has been the prominent sign. The paroxysmal hypertension so characteristic of the disease in the adult is seldom seen. In the 24 cases in children reviewed by Moore *et al.*[2] only one showed paroxysmal hypertension. Symptoms produced by the tumour are usually related to the excessive production of adrenaline or noradrenaline. Apart from the hypertension, prominent symptoms are headaches, abdominal pain, sweating with cold extremities, palpitations, and polydipsia. Raised basal metabolic rate, hypertensive retinopathy, loss of weight and albuminuria are also common features.[1]

The tumour itself may produce manifestations that are sometimes misleading. In Cone's case[5,7] the second tumour presented as a swelling in the neck, with Horner's syndrome. In the following case report the patient presented with an unexplained pyrexia.

CASE REPORT

E.N., a 10-year-old Indian boy, was admitted to the Red Cross War Memorial Children's Hospital for the first time in June 1958. He complained of headache, fever and abdominal pain for 1 month, and there was loss of weight, dizziness and sweating attacks.

He was a thin boy weighing 48 lb. The respiratory, nervous and cardiovascular systems were normal. Temperature 102°F. No abnormal blood pressure noted. Hb. 7·2 g.%; w.b.c. 14,600 per c.mm. (polymorphs. 92%, lymphocytes 6%, mononuclears 2%); ESR 160 mm. in first hour, Westergren. Mantoux positive 1/1000. X-ray of chest and abdomen, intravenous pyelography, agglutination reactions, Wasserman reaction, ECG, and bacteriology of stools and urine, were all normal. Serum protein—total 7·76 g.% (albumin 2·5, globulin 5·2).

Because of the positive Mantoux reaction, fever, and raised erythrocyte sedimentation rate a diagnosis of tuberculosis was made, though the site of the lesion was not apparent. The boy was given isoniazid and para-aminosalicylate and the temperature and sedimentation rate fell to normal. He was kept on this therapy for 6 months, when it was discontinued. He then became ill again with a recurrence of all his previous symptoms.

In February 1959 he was readmitted. This time his weight was 54 lb., his temperature 100·2° F and he was sweating profusely. The urine showed a trace of albumin. Serum albumin 3·21 g.%, globulin 5·17. All the previous tests were repeated and confirmed. In addition, X-ray of the spine and skull, and air encephalograms, were normal. The ESR was 136 mm. Westergren.

He was treated with streptomycin and isoniazid once more and, because of his severe headache, a lumbar puncture and air encephalograms were done. The cerebrospinal fluid showed a protein of 200 mg.% (globulin ++), but was otherwise normal. Following this investigation he complained of headache and sweated profusely. Six hours later he developed right-sided convulsions (B.P. 130/90 mm. Hg), from which he completely recovered in 24 hours. The CSF was normal 2 weeks later and the temperature had by then returned to normal. He continued to have symptoms, however, but was discharged, to be followed up as an out-patient. The elevated blood pressure was not investigated further.

In August 1959 he was admitted again for reappraisal. The same examination was made as before. The temperature was 99° F but it settled after 24 hours and remained normal without specific treatment. The ESR was 145 mm. Westergren and the blood pressure 135/90 mm. Hg. The urine on occasion showed a trace of albumin. Serum albumin 2·77 g.%, globulin 5·15, gamma globulin 1·03. Muscle biopsy was done and L.E. cells looked for in the peripheral blood because disseminated lupus erythematosus or polyarteritis nodosa was suspected. There was no evidence of a collagenosis, however.

The patient always perspired excessively, but this was attributed to a tuberculous infection. Because of the raised blood-pressure reading and albuminuria, the blood pressure was measured daily and the urine tested. It was thus found that on occasion the blood pressure would be 150/130 mm. Hg (see below), and sweating was possibly more marked at such times, though the correlation was not at all dramatic. At other times the blood pressure was 130/90. Because of the paroxysmal hypertension a diagnosis of phaeochromocytoma was suspected. No significant association between the raised blood pressure and the hyperidrosis was considered until that suspicion arose. Urine specimens were therefore tested for catecholamines. The positive results of the test and the method

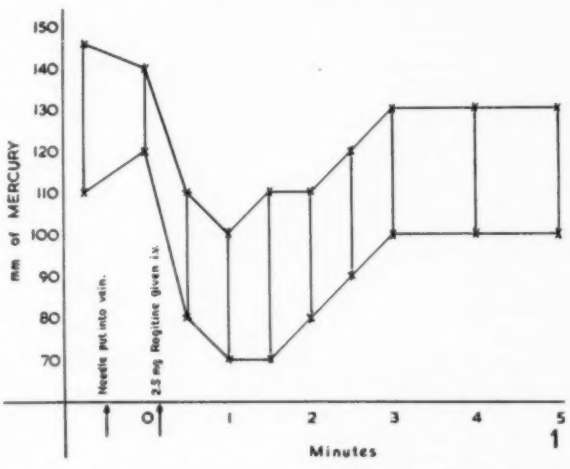

Fig. 1. Case E.N. Showing reaction of blood pressure to phentolamine

employed are fully discussed elsewhere* by Professor Sapeika.[9] Because of his findings a phentolamine (rogitine) test was performed as described by Gifford *et al.*[10] The result was positive. An immediate drop in blood pressure followed an intravenous injection of 2·5 mg. of phentolamine (Fig. 1). On these grounds a laparotomy was performed under general anaesthesia, the surgeon electing to dispense with any attempt at localization. No specific pre-operation therapy was given.

Operation

Before the operation was begun the right brachial artery was canalized through a small incision and a polythene catheter inserted into the artery. The patient was then connected to a 2-channel electrocardiograph and pressure-recording apparatus. Throughout the operation continuous blood-pressure and ECG monitoring was thus possible. An intravenous drip infusion of 5% dextrose was started in the other arm. A 2-way tap on this circuit enabled immediate intravenous injections to be given as required. A transverse upper abdominal incision was used. At the commencement of the operation the blood pressure was 150/110 mm. Hg. The left adrenal area was explored and a large tumour found and removed. With the handling of the tumour there was a sharp rise in blood pressure to 220/170 which was well controlled by 2·5 mg. of phentolamine, the pressure falling to 140/90 in 1½ minutes. Later there was a slow rise again to 215/197. The other side was explored and another tumour found. This was then also removed. The whole of the left adrenal was removed, but only a piece of the right adrenal. With the removal of the second tumour there was a sharp fall in blood pressure to 60/50. A noradrenaline drip was commenced, 4 mg. of 'levophed', dissolved in 200 c.c. of 5% dextrose, being given intravenously as well as 100 mg. of 'solucortef' (hydrocortisone sodium succinate). The blood pressure returned to 90/80 and 15 minutes later was 110/75. Post-operatively, hydrocortisone was given intramuscularly in decreasing amounts for the next 7 days. Initially 100 mg. of hydrocortisone was given 8-hourly for the first 16 hours after the operation. Thereafter 50 mg. was given 8-hourly for 24 hours, gradually reducing the dose by 25 mg. a day.

During the immediate post-operative period a continuous noradrenaline drip was maintained. For the first 10 hours 4 mg. of 'levophed' (noradrenaline bitartrate) dissolved in 200 c.c. of 5% dextrose was given. The systolic blood pressure was maintained at between 110 and 120 mm. Hg. For the next 10 hours, 3 mg. was given in 200 c.c. of 5% dextrose and then 2 mg. in 200 c.c. of 5% dextrose over 5 hours. As the blood pressure remained constant the 'levophed' was decreased to 1 mg. in 200 c.c. of Darrow's solution over the next 9 hours, and then to 0·5 mg. for the next 7 hours. The systolic blood pressure remained at 120 mm. Hg, and so the drip was discontinued 41 hours after the operation. No further fall occurred. During this time no fluids were given by mouth.

Pathology Report (Dr. L. Anstey)

Specimens consist of two circular tumours. The left weighed 79·6 g. and measured 7×5×4½ cm. The adrenal gland capped the tumour. On cut section the periphery of the tumour was fleshy and pink in colour, while in the centre there was a yellowish area of necrosis 3 cm. in diameter. In this was an area of haemorrhage 1 cm. in diameter.

The right tumour weighed 38·4 g. and measured 4½×2½×5 cm. On cut section the tumour was uniformly fleshy, with one small area of haemorrhagic discoloration. There was a small tag of tissue at one pole, but this did not appear to be adrenal.

Histology. Both tumours consisted of groups of large cells surrounded by narrow trabeculae of connective tissue within which thin-walled blood vessels were discernible. There were large areas of haemorrhage and necrosis in both tumours.

Comment. The features of both tumours are those of phaeochromocytoma. There was no evidence of malignancy in the sections examined.

Follow-up

The patient has subsequently been seen 5 months after operation. The blood pressure remains normal and he has gained 9 lb. in weight. There was a recurrence of his pyrexia and the raised blood sedimentation rate, but this has now returned to normal. The reversal of the albumin-globulin ratio is still present. Post-operative urine examination revealed no catecholamines.

* See page 257 of this issue.

DISCUSSION

Some of the features presented in this boy were quite typical of the disease in children described by previous authors and now well documented. The majority of cases, however, have shown persistent hypertension and their symptoms were usually related to this. Bilateral tumours are not common and this is the first case reported from this country in which multiple tumours have occurred in a child under the age of 14 years.

This patient presented some interesting features that were not fully elucidated. These were the raised sedimentation rate, fever, and reversal of albumin-globulin ratio. Considering, however, that both tumours had areas of haemorrhagic necrosis in them, it is possible that this was responsible for the fever and increased ESR. These are certainly well-recognized features of necrosis occurring in tumours in adults. There was, however, in the strongly positive Mantoux, evidence of an infective aetiology, and initially the expected response to anti-tuberculous therapy gave no reason to suspect any other pathology. It was only later, when investigation of a suspected intracranial tuberculoma produced collapse, that attention was drawn to the blood pressure. It was then realized that an earlier recording of the blood pressure had been overlooked. From this point onwards the investigations were diverted into an entirely different channel and it quickly became apparent that a phaeochromocytoma was probably present.

It was intended to try to obtain further evidence, by pyelograms and retroperitoneal air studies, of suprarenal abnormality but, in view of the upset which had followed encephalography and the inconclusiveness of these procedures, the surgeon decided to explore the abdomen without further delay.

The episode associated with the air encephalogram was probably due to hypertensive encephalopathy. The reversal of the albumin-globulin ratio remains unexplained. As there was persistence of this reversal 5 months after operation, it is postulated that the patient must still have some illness that has not been elucidated.

Management

It is advised by Daeschner *et al.*,[1] Robinson *et al.*[3] and others, that pre-operative phentolamine should be used to control the hypertension. This is probably not necessary when the hypertension is paroxysmal and was not done in this case.

Once the diagnosis has been made, it is unnecessary to delay operation. Localization of the tumour by presacral insufflation has been recorded on a number of occasions. It is not very reliable and is also unnecessary. Wide laparotomy should be the rule, especially as 20% are cases with multiple tumours[11] and the abdomen will always have to be explored. An approach to the kidney through the loin has often proved unsatisfactory for this reason.

Constant blood-pressure monitoring with a 2 channel recording apparatus was extremely helpful during the operation. Changes in cardiac rhythm and blood pressure could be detected immediately and remedied by appropriate measures. The readings are accurate and instantaneous and the continuous record is incomparably superior to the use of a sphygmomanometer. The mortality rate from the condition has been high: Moore *et al.*[2] recorded 13 deaths out of the 24 cases which they reviewed. It is probable that more careful

management of blood pressure during the operation may help to prevent this in the future.

The diagnosis of a functionally active phaeochromocytoma depends on two physiological approaches. These are (1) the use of adrenolytic agents such as phentolamine or benzodioxane, and (2) the determination of catecholamines in the blood or urine. The value of testing for catecholamines in the urine of the patient was well demonstrated. Neither the phentolamine[10] nor the benzodioxane[12] test is infallible and both sometimes give false negative results. In this patient the response to phentolamine as a test and also during operation was very good. Provocative tests with histamine are only of value in a patient with paroxysmal hypertension, but are dangerous. The demonstration of urinary catecholamines remains a most satisfactory investigation in the diagnosis of phaeochromocytoma.

SUMMARY

1. The increasing number of cases of phaeochromocytoma reported in childhood is indicated. With this present one, 43 cases have been recorded in the literature.

2. A detailed history of an unusual case of phaeochromocytoma is given.

3. Continuous direct blood-pressure and electrocardiographic monitoring is advised as a satisfactory means of keeping a check on the patient's condition during operation.

4. The value of testing the urine for catecholamines in the presence of unexplained hypertension is reaffirmed.

I should like to thank Dr. J. W. F. Mostert, Superintendent of the Red Cross War Memorial Children's Hospital, for his permission to publish this case, Prof. F. Ford for his help, Prof. J. H. Louw, who performed the operation, and Dr. L. Anstey for the pathology report.

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PHAEOCHROMOCYTOMA: LABORATORY DIAGNOSIS

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Phaeochromocytomata may arise in the adrenal glands or in extra-adrenal medullary tissue. Although the tumour is responsible for less than 1% of all cases of arterial hypertension, its early and accurate diagnosis is important because it is a cause of hypertension that is curable by surgery.[1-3] The hypertension may be sustained, but in 30% of cases it is paroxysmal in type. Hypertension is not invariably present, and tumours may occur without clinical evidence of hormone production.

Phaeochromocytomata can be detected from clinical data in about 50% of cases, but special tests are necessary to establish the diagnosis. The tests are of two kinds,[3, 4] viz. clinical pharmacological tests and laboratory examination of the urine for catecholamines or other excretory products.

The clinical tests that may be performed on the patient are (*a*) provocative tests, in which histamine or methacholine is used to stimulate the tumour to produce a paroxysm of hypertension, and (*b*) adrenergic blockade tests, in which phentolamine (regitine) is administered to antagonize the action of circulating adrenaline and noradrenaline. The use of these drugs requires very careful observation of the proper indications and the details of technique. It must also be appreciated that false positive and false negative responses may occur.

LABORATORY TESTS

Estimations of catecholamines or other adrenal medullary substances in the urine are a more reliable guide to the presence of adrenal medullary tumour than the clinical pharmacological tests. Various chemical, physical and biological methods are available for the measurement of adrenaline, noradrenaline, and related compounds in the urine.[3,5,10,11] The tumour tissue can also be assayed; large amounts of noradrenaline and adrenaline have been demonstrated in these tumours.[5-9] The proportion of the two catecholamines excreted in the urine and the relative content of these substances in the tumour is well correlated.[12]

Most laboratory tests of the urine for this purpose are relatively difficult and time-consuming and therefore unsuitable for routine screening of a large number of urine samples. A fluorimetric test studied in this laboratory[13] was found to be less reliable for the screening of phaeochromocytoma than the 'cat' test described below. The recently published methods[10, 11] for quantitative estimation of 3-methoxy-4 hydroxymandelic acid are not suitable for routine screening of urine samples. In special cases they would be of inestimable value, since they determine the amount of this major metabolite of adrenaline and noradrenaline, which is of great diagnostic importance; large amounts may be excreted in the urine of patients with phaeochromocytoma.

The simple method of biological assay described by Moulton and Willoughby[14] is useful as a screening and diagnostic test. Untreated urine is examined for pressor activity in the anaesthetized cat. Random samples of urine or aliquots of 24-hour specimens are required; 50 mg. of ascorbic acid is added to each sample as anti-oxidant, and the samples are stored at 0°C until the test can be performed. The cat is prepared for recording arterial blood pressure and for femoral intravenous injections. Certain modifications of the test procedure have been recommended by the original authors (personal communication). Thus, a ganglion-blocking agent such as hexamethonium bitartrate, about 25 mg., is given initially to lower the blood pressure and to sensitize the preparation for pressor responses. The pH of the urine samples is not adjusted initially, but only if a sample produces a significant rise in blood pressure. The standard dose (0·2 microgram in 1 ml.) of noradrenaline and of

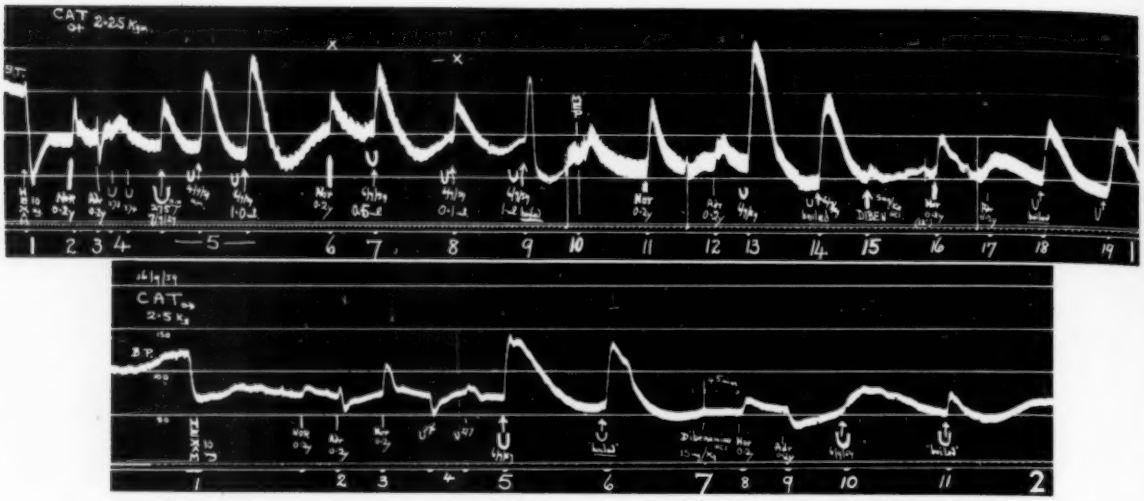

Fig. 1. Marked pressor action of 1 ml. of urine: at 5 and 7 (compared with 0·2 microgram of noradrenaline at 2 and 6); at 13, compared with noradrenaline at 11, after mepyramine given at 10; all effects reduced after dibenamine given at 15. Note: 0·1 ml. of urine at 8 produced an effect similar to that of noradrenaline at 6. At 18, 'boiled' urine still produced a marked pressor action.

Fig. 2. Marked pressor action of urine at 5, and of 'boiled' urine at 6, compared with noradrenaline at 3; all effects much reduced after dibenamine given at 7.

adrenaline is given initially and at suitable intervals during the test. All injections are given intravenously through the cannula in the femoral vein in constant volume (1 ml. of drug solution or 1 ml. of urine), each dose washed in at constant speed with a constant volume of saline (2 ml.). If when the urine is injected an alteration in blood pressure occurs equal to or greater than that produced by the standard dose of noradrenaline or adrenaline, a study is made of the modifying effect of an antihistamine drug and an adrenergic blocking drug on the vasomotor response. The heating of a suspect urine with sodium hydroxide and ferric chloride is no longer part of the procedure.

Of 330 samples of urine examined in this laboratory, evidence for the diagnosis of phaeochromocytoma has been found in 3 cases. Details of the clinical findings in the last of these cases are reported elsewhere* by Dr. Rainier-Pope.[15] The present note deals with the bio-assay findings in this case.

CASE REPORT

Samples of urine from an Indian boy E.N., aged 10 years, were examined for pressor activity. As shown in Figs. 1 and 2 remarkable increase in the blood pressure was produced by a number of different urine samples. In one instance 0·1 ml. urine produced an effect equal to that produced by the standard dose of noradrenaline. Incidentally the pressor activity of the urine was not abolished by oxidation with ferric chloride. The effects produced by the adrenergic drugs and the urines were more marked after the antihistaminic drug (mepyramine) (Fig. 1), and were much reduced after the adrenergic blocking agent (dibenamine) (Figs. 1 and 2).

An aqueous extract prepared from the tumour tissue ground with sand was found to exert marked activity; 1 g. of tissue contained pressor activity equivalent approximately to 4 mg. of noradrenaline. (Some workers have extracted tumour tissue ground with sand and 10 ml. of N/10 HCl per g. of tumour, heating in a boiling water bath for 10 minutes, filtering, and adjusting the filtrate so that 1 ml.=0·1 g. of tumour.)

* See page 255 of this issue.

Samples of urine collected on 3 consecutive days in the post-operative period showed no pressor activity.

DISCUSSION

It is advisable to perform laboratory tests in all cases of suspected phaeochromocytoma before exploratory surgery. The 'cat' test for catecholamines is simple, and many samples of urine may easily be examined at one session. Although an increase in the excretion of catecholamines has been demonstrated in the majority of cases of phaeochromocytoma when the patient's blood pressure is elevated, an increase has occasionally also been demonstrated even when the blood pressure was normal at the time of collection of the urine. It must be noted that in the bio-assay of urine and of tumour extract the responses obtained are the resultant of competition between noradrenaline and adrenaline for equal cell receptors, and interference in their actions sometimes occurs. The pressor activity of the urine may also be modified by other substances that are present, so that small but significant increases in catecholamine excretion may therefore be missed. The standard doses of noradrenaline and adrenaline given singly produce effects that may be different from a urine sample because of their separate unimpeded action.

As an approximate guide to the total amount of noradrenaline that may be found in the urine in 24 hours the following values have been presented by Goldenberg:[16] 0 – 50 micrograms in normal subjects, 0 – 100 in essential hypertension, and over 100 in patients with phaeochromocytoma (usually 600 – 2,700 in persistent hypertension and 190 – 1,530 in paroxysmal hypertension). Sjoerdsma[5] states that the normal urinary excretion of adrenaline plus noradrenaline is usually less than 100 micrograms per day, whereas in phaeochromocytoma it is typically in the range 300 – 3,000 micrograms per day; most of the tumours contain 500 – 10,000 micrograms

per g. of total catecholamines. According to Robson and Keele[3] the total amount of the two amines in tumours is 5 - 15 mg. per g. (in normal human adrenal tissues about 1 mg. per g. of pressor substances).

SUMMARY

The simple screening 'cat' (blood pressure) test for detecting catecholamines in the urine of patients with a suspected phaeochromocytoma has a number of advantages. The cat preparation is easily set up, it is reliable, numerous urine samples can be investigated at one session, and samples can be repeatedly examined under fairly exact conditions. Some aspects of the technique of the assay and a positive result are reported. References are given to important general review articles on phaeochromocytoma.

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INTRATHORACIC DUPLICATIONS OF THE FOREGUT

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PART II

CLINICAL IMPLICATIONS

Diagnosis

Awareness of duplication is the prerequisite to correct diagnosis. Chest radiographs of all patients presenting with unexplained mediastinal masses should be carefully examined for spinal deformities. All infants and children with swellings of the mediastinum of unknown origin should have antero-posterior and lateral films of the vertebral column, including the cervical spine. The finding of spina bifida, fused cervico-dorsal vertebrae and other associated congenital spinal deformities should immediately suggest the possibility of foregut or intestinal duplications. When a vertebral lesion co-exists with a mediastinal duplication, especially of the elongated type, the co-existence of an intestinal duplication becomes probable. When the subject in addition loses blood in the stool and complains of vague, periodic colicky pains in the abdomen, this association becomes a certainty. Endoscopy and bronchograms are seldom helpful but a barium swallow is useful and should always be done.

Complications

Symptoms may be absent, slight, or so severe as to demand emergency measures. Duplications of bronchial origin seldom give rise to serious complications in the adult but may cause compression and thus give rise to respiratory distress, dysphagia or bone erosion, which might be particularly dangerous in infants. On the other hand duplications of alimentary origin are potentially extremely dangerous, especially in infants and children, where mild warning symptoms may either be unnoticed or wrongly interpreted. Complications are more the rule than the exception,[36] and fatal termination is not uncommon[72,69] (case 5 in this series). Active peptic ulceration is common; it may lead to haemorrhage and perforation with all its concomitant sequelae. The inflammatory reaction around the ulceration is most intense and may result in dense adhesions to neighbouring structures. Fistulae may form into the bronchus,[31] the lung,[50] the oesophagus,[37] and the pericardium (case 5). The cysts may become infected, or may cause obstruction, either by predisposing to volvulus[42] or by encroaching on the lumen of the gut.[67] Dohn *et al.*[30] found that 60% of the subjects with enterogenous cysts which they reviewed in 1952 had intestinal obstruction, and 25% of the total were fatal.

Treatment

Because of the danger of complications and the uncertainty in the diagnosis there is no place for conservative treatment in suspected duplications of the foregut. Anaesthesia and thoracic surgery are now sufficiently advanced to urge surgical extirpation without hesitation in any age-group. The diaphragm should be incised and the abdomen explored if there is any indication of a prolongation downwards. If the cyst wall forms part of the membranous trachea, the mucosa only should be removed here and the muscle left behind.

THEORIES ON THE ORIGIN OF DUPLICATIONS

The multiple theories that have been advanced to explain duplications on an embryological basis are sufficient evidence of their complexity. From the above case histories and comments it is clear that the different types of foregut duplications sometimes merge imperceptibly into one another. Furthermore the relationship between mediastinal and abdominal duplications and other anomalies is so constant and similar that a theory cannot be accepted if it fails to take into account all the variables. Finally the common characteristics of certain congenital syndromes and abnormalities suggest a common denominator. Theories should be critically examined in this light.

1. *Vitelline Theory*

Since Meckel's original description of the intestinal diverticulum which still carries his name, this vitelline vestige has been included in the list of possible causes of intestinal duplications. It is hardly necessary to point out that in Meckel's diverticulum there is a 5 to 1 sexual preponderance of males;[15] that associated anomalies are not commoner than in the average population, and that it is difficult to understand how duplications in the oesophagus and rectum can arise from this source. Further, Edwards[21] reported a typical Meckel's diverticulum in a patient with a duplication and quite rightly concluded that two diverticula of Meckel

do not occur in the same bowel. This view is now only of historic interest. Black *et al.*[5] regarded intrathoracic duplications as vestigial intrathoracic remains of vitelline veins. However, at the time of obliteration of the vitelline duct, the vessels of the dorsal mesentery are well developed, and these cysts should be vascularized by mesenteric vessels, which is difficult to imagine when they occur in the rectum or at the base of the tongue.

2. *Theory of Diverticulation*

Lewis and Thyng[49] found that knob-like diverticula are commonly present in the foetal alimentary tract of pigs, rabbits and man. These normally regress, but the authors suggested that persistance or sequestration might give rise to duplication. The fact that duplications are most commonly found in the terminal ileum where foetal diverticula preponderate has been used further to strengthen this argument.

Confusion is caused by the inappropriate use of the loose term 'diverticulum'. Meckel's diverticulum for instance is embryologically and pathologically not cognate with the embryonal out-pocketing seen by Lewis and Thyng.[49] Yet abnormal differentiation of epithelium, such a characteristic feature in duplications, is not uncommon in a Meckel. Congenital diverticula of the alimentary tract are usually more or less of the same size, are known to preponderate in certain areas of the gut, are often multiple, and share the same epithelial and muscular layers as the parent organ at that level. Congenital diverticula should also be differentiated from the acquired type of pulsion diverticulum (commonly at the crico-pharyngeus and lower end of the oesophagus), the traction diverticulum (at the hilum of the lung), and those that develop along the path of larger blood vessels, especially in the colon and small intestine. Even some of these so-called acquired diverticula may be congenital, characterized by the same features as duplications. For instance, Baar *et al.*[3] described a case of epiphrenal oesophageal diverticulum containing the normal layers of the oesophagus but connected to the lung by a band of functioning pancreatic tissue, which proved its congenital origin. There is no evidence that diverticulation occurs in the developing oesophagus, and there is certainly no association with spinal deformities, as there is in duplication of oesophageal origin. Whereas it is possible that congenital diverticula of the gut develop as a persistance of the above-mentioned epithelial buds,[49] it is hardly conceivable that these simple, uniform aberrations would grow and develop into complex duplications anywhere from the pharynx to the anus. All the facts suggest two entirely different entities, if not in origin then at least as end results.

3. *Theory of Vacuolization*

Shallow[73] and Keith[40] felt that duplication results from failure of, or abnormal coalescence of vacuoles, and Bremer[12] still blames abnormal vacuolization for all duplications. He supports Lewis and Thyng's theory that diverticula develop as minute hollow buds from the epithelial lining of the gut, which grow into the subepithelial connective tissue in embryos of 20 - 30 mm. Most of these disappear, but some persist and may with development push through the outer layers as a pedicled diverticulum or close off as a free cyst. Duplications, he holds, are formed at the time of epithelial proliferation and rapid elongation of the gut, and are due to persisting vacuoles which fail to communicate with the parent lumen.

Contrary to a misconception which still lingers, I find that the lumen of the oesophagus is never completely occluded, but its size varies considerably at different levels and during different stages of development. Before the embryo reaches 10 mm. the lumen is already established; vacuoles only appear by about 12 mm.[7,8] The caudal end of the oesophagus elongates rapidly and is by far the narrowest part, a feature which probably led to the belief[44] that the lumen becomes occluded by epithelial proliferation and later re-established by vacuolization, a process which is well known to occur in the oesophagus of certain lower vertebrates.[58] After a study of serial sections through the oesophagus in embryos varying from 12 to 14 mm., I was never satisfied that the confluence of vacuoles, which in some areas was completely absent, contributed substantially to the lumen. At 29 mm., when the vacuoles were most conspicuous, the lumen was already relatively large, and although vacuolization continues till about 75 mm., in not one instance was a single vacuole seen outside the developing muscularis mucosae. Even Bremer could not explain complete duplication of the stomach on the basis of abnormal confluence of vacuoles, and postulated that this type of duplication was due to fusion of opposing walls. It is equally difficult to believe that vacuoles can give rise to a double colon with two perfectly formed separate appendices, or a second oesophagus which stretches from the root of the neck to the 12th thoracic vertebra through an accessory diaphragmatic hiatus.[48] The main reason why vacuoles cannot possibly be a causative factor for elongated duplications is that they appear too late in the embryo to derange certain fundamental embryological developments. Thus, spinal abnormalities cannot be explained on such a hypothesis.

4. *Theory of Imperfect Separation of the Tracheopulmonary Bud from the Oesophagus*

The theory of Olenik *et al.*[59] that all cysts are derived from the foregut at or near the origin of the lung buds might explain some mediastinal duplications but fails to account for those which occur elsewhere. The same objection may be raised against Bergmann *et al.*,[4] who thought it apparent that tracheobronchial rests in the oesophagus as well as tracheo-oesophageal fistula are the results of failure of the normal embryonal process of separation of the respiratory tract from the primitive oesophagus. The predominance of simple bronchogenic cysts in the superior mediastinum and their common intimate connection with the tracheobronchial system suggests that many of them might arise at the site of separation. That this may not necessarily be a simple local process is shown by the co-existence of vertebral anomalies in cases with bronchogenic cysts.[77,52] In the present series some of the most typical bronchogenic cysts had no evidence of cartilage in their walls, and the case of bronchogenic cyst with an associated spinal deformity was very similar to the cases quoted by Tucker[77] and Maier.[62] The association of bronchogenic cysts with other anomalies such as cryptorchidism,[39] patent foramen ovale and ductus arteriosus,[28] ductus arteriosus in twins,[16] and congenital cataracts,[19] further suggests that a more generalized embryological disturbance exists. A mere failure of normal separation of the respiratory and alimentary tracts would similarly not explain the common co-existence of tracheo-oesophageal fistula with congenital heart disease, malformation of anus and rectum, malrotation of the gut, abnormalities of the kidney, and atresia of the small intestine. As in duplications, vertebral anomalies, pulmonary aplasia and concomitant intestinal duplications may occur in tracheo-oesophageal fistula. This intrinsic tendency to widespread malformation was admirably illustrated by a case of tracheo-oesophageal fistula in which there were also an imperforate anus and an accessory thumb on the right hand.

5. *Sequestration of Embryonic Multipotential Cells of the Primitive Alimentary Anlage*

A full discussion of the many fascinating facets of sequestration unfortunately falls outside the scope of this paper. I shall only briefly point out the relationship between 'extralobar sequestration' of the lung, of which there are 2 examples in this series, and duplication of the foregut.

The respiratory system arises from a median longitudinal groove in the ventral wall of the pharynx when the embryo is about 3 mm. long. This ventral outgrowth is soon pinched off from the foregut by lateral constrictions, which start caudally and proceed headwards. My own observations support Boyden's view[11] that the primordial trachea then grows caudally, becomes evaginated, and divides into 2 buds which rapidly extend into the surrounding mesenchyme with further branching to form the lobar and segmental bronchi. A disturbance of normal separation may therefore be responsible for a variety of the well-known tracheo-oesophageal defects, or separation may be apparently normal but duplications may form either from the oesophagus proper or the primitive tracheobronchial system. Disorganized growth may also occur later and therefore be evident in a more peripheral part of the bronchial tree. The most minor disturbance occurs when additional buds develop in an otherwise normally developed bronchial system (supernumerary segment) or when the segmental buds are normal numerically but in the wrong position (misplaced segments). These may persist, as is commonly seen in the right upper lobe, and develop as normal fully-functioning units in abnormal positions, served by a patent bronchus and supplied by a branch of the pulmonary artery. On the other hand, these components may degenerate and finally disappear. Small buds of the dissociated tissue may persist and give rise to unilocular or multilocular cysts, which may or may not retain

their connections with the bronchi and may even be wrongly reported as teratomas.[39,38] If abnormal growth occurs even more distally, solitary intrapulmonary cysts may arise or cystic masses of disorganized lung tissue may be formed, with or without systemic pulmonary arteries, known as intralobar sequestrations. These may start to develop *in utero* but, if so, then it is surprising that not a single case was found under 1 year old in over 16,000 necropsies done at different centres.[63,9] However, it does not mean that the condition cannot occur, and one reason why it could be missed easily at necropsy is that the pathological anatomy might be so nearly normal that it escapes notice. As budding might continue for a further 7 generations after birth (18 before birth[34]) the possibility exists that this abnormality might arise or be accentuated in postnatal life. Abbey Smith[74] postulates that when such a segment is supplied by a systemic pulmonary artery and subjected to the high systemic blood pressure cystic changes and fibrous degeneration take place. He could not explain one of his own cases with a 'whole lobe type of lesion' on this theory and, although his theory is attractive and simple, it does not explain why mature individuals with single stout systemic pulmonary arteries[64,65,54] or multiple systemic arteries (5 - 6)[33] do not develop cysts in the segments supplied by those vessels. Finally, Boyden[10] has shown cysts without evidence of systemic vessels and a systemic pulmonary artery without any evidence of intralobar sequestration in 2 embryos of 31 and 41 mm. respectively.

Lung tissue may not only be developed from the conventional source, but also from any other part of the primitive foregut. Again the primary disturbance of growth may be minimal so that solitary cysts may develop within the wall of the oesophagus or outside this organ and contain all the characteristics of a foregut duplication of bronchial origin. If the disturbance in growth is more pronounced, accessory lung tissue may form, known as extralobar sequestration, which may or may not be associated with an ordinary duplication of oesophageal origin. Two cases in the present series are examples of this type. When the connection of the sequestration to the foregut persists it forms irrefutable proof of the origin of the anomaly. Connections to the stomach[71] and the oesophagus at bifurcation level[25] have been described. The extralobar sequestrations are usually supplied by one or more systemic arteries arising directly from the aorta, intercostals or phrenic arteries. These may even reach the size of the aorta.[58] In another case (adult female) that was shown to me, the systemic artery arising from the phrenic was the size of a pencil and supplied a right posterior lower-lobe sequestration. After operation the systemic hypertention dropped to a steady 140 mm. Hg systolic. Weisel *et al.*[51] considered sequestration as bronchogenic cysts with associated anomalous vascular supplies. Cole *et al.*[17] regarded bronchogenic cysts, anomalous systemic vessels, extralobar sequestrations, and diaphragmatic defects, as related conditions with a common developmental error. Caffey[12] included diaphragmatic defects, cardiac and renal malformations, skeletal abnormalities, and absence of the vagus nerve as commonly associated with aplasia of the lung, and further considered the high frequency of association of pulmonary aplasia with hemivertebra to justify the combination as a syndrome. The high preponderance of left-sided lesions (90%[78]) are explained by Bolck[6] on the basis of late closure of the left leaf of the diaphragm, which would also account for the high incidence of diaphragmatic defects.

The theory[64] that both intralobar and extralobar sequestrations are due to traction on one or more buds of the developing bronchial tree by abberrant arteries, while the lung is undergoing developmental shifts, cannot be accepted embryologically. Even in the early stages the bulbous tips are well developed and contain a thick pseudocolumnar epithelium with several rows of cells, while the vascular network is still either non-existent or essentially capillary (Boyden's 'no man's land'[10]) and cannot possibly 'capture' such a stout bronchial bud. There is also no evidence to support the idea that the pulmonary and systemic arteries are competing to capture the lung buds. It seems much more likely that aberrant vessels are persistent branches of the vascular plexus which surrounds the visceral tubes very early in development. When such a channel exists it merely takes over the bud (later to become duplication of bronchial origin or sequestration) in its vicinity and may eventually be moved apart as growth takes place. The differences between intralobar and extralobar sequestrations have been admirably covered by Abbey Smith,[74] and need no further elaboration. The intimate co-existence of an elongated duplication and an extralobar sequestration with systemic arteries (case 5) is beyond doubt.

6. *The Notochordal Theory*

Feller *et al.*[24] (1929) collected 28 cases of malformations which involved both the central nervous system and the alimentary canal; every case also had anterior spina bifida. Puussepp[66] was the first to describe a foregut duplication with the characteristics of small intestine within the vertebral canal; it was lying within the dura dorsal to the cervical spinal cord (C3) in a man of 27 years. Guillery's case[32] is referred to above. Knight *et al.*[43] reported a large duplication characteristic of stomach which was lying within the cord in the cervicodorsal area of a child of 15 months. Recently Harriman[35] reported another 'enterogenous cyst' within the spinal cord at vertebra D3; there were gross vertebral anomalies from C7 to D7. Unlike teratomas, which contain only ectodermal and mesodermal elements, this cyst contained mesodermal and endodermal elements, which were surprisingly well differentiated and orderly. This suggested that the ectopic cyst was extruded into the spinal canal through the anterior spina bifida and might or might not be related to a corresponding tumour in the mediastinum, of which no evidence could be found.

The significance of vertebral anomalies was first noticed by Stoeckel.[75] Veeneklaas[70] suggested that imperfect separation of the notochord from the endoderm may lead to withdrawal of a pouch from the primitive alimentary tube into the mesodermal anlage of the vertebral column, which may then develop into a duplication with or without communication with the digestive tract, while the attachment of the pouch to the notochord may interfere with the normal development of the spine. Fallon *et al.*[23] gives a good account of the notochordal theory and feels that all duplications above and below the diaphragm as well as the various forms of vertebral dysplasia are the result of upset of the stage of excalation of the notochord.

From what has been said before it should be clear that spinal abnormalities usually do not occur in duplications of the lower intestinal tract, although these may be extensive, and further that vertebral anomalies occur typically with certain types of malformation without any apparent involvement of the alimentary tract. I therefore suggest that the primary defect does not lie in the notochord, but that the skeletal and nervous dysplasias are part of the greater or lesser disturbance in development which might affect general systems of the embryo at various levels in many different ways.

7. *Theory of Twinning*

This theory has been rejected in the past but recent work[16] has shown that twin formation may well occur, a theory which would appeal particularly in some of the elongated duplications of the large bowel. Morton[56] published a case of duplication of the pituitary and stomatodaeal structures in a 38-week male infant. In order to explain such a duplication or 'twinning' it is necessary to envisage a stage in the early development during which a small deviation from normal will initiate a chain of events that will bring about the ultimate abnormality in all its variations.

8. *The Imbalance of Organizational Substances in Embryonic Life*

Needham[57] favours 'morphogenetic hormones or stimulating substances' as being important factors in 'many inheritable aberrations or modifications of development.' Baar *et al.*,[3] in their series, explained all duplications and deviations of the foregut 'on the basis of an interference with organizer centres leading to a failure of resorption of accessory buds of the primitive foregut and the displacement of the growing buds within intersections of morphogenetic gradients resulting in a preponderance of self-differentiation over dependent differentiation. . . .'

The hormonal theory has the beauty of simplicity. In my opinion it is the only logical concept that is compatible with all the facts, and the following hypothesis is put forward on this basis:

Localized derangements in embryonal metabolism and hormonal guidance may result in abnormal development of normal tissues. The severity and the time of the upset decides the position, type and extent of the ultimate abnormality. Minor disturbances may be rectified by the organism without

any subsequent trace, or anomalous developments may be incorporated as normal functional units (supernumerary and misplaced bronchi). Localized cysts may also develop at different levels from the tracheobronchial tree or oesophagus, with the characteristic features of the parent organ but no associated anomaly. Abnormal growth may be early and give rise to extralobar sequestration, or possibly late and lead to intralobar sequestration, with or without concomitant congenital vascular anomalies. As a result of one anomaly other disturbances in growth may follow which are not primarily hormonal but mechanical, and such permutations may present bizarre patterns which are difficult to explain. If the disturbance is more severe, round duplications may result which retain their endodermal competence, and because they are out of position and disorganized the histogenic differentiation is less complete and precise. The varied mucosal cell type is merely a reflection of the wide developmental potential of endodermal tissue.

With even more severe or persistent disturbances elongated duplications result which represent the anatomy as well as the physiology of the parent organ to a surprising extent. The upset in local hormonal control spreads to neighbouring or associated organizer centres and high spinal deformities become the rule rather than the exception. Concomitant abdominal duplications and malrotation of the gut now occur, and when the oesophagus is completely duplicated the spinal deformity even involves the sacrum. A less localized disturbance may involve several systems and give rise to typical and well-known syndromes. Minor generalized upsets, or localized upsets occurring at different times, may give rise to apparently unconnected congenital anomalies, while severe hormonal imbalance results in frank monster formation.

Töndury,[78] who reviewed developmental physiology of abnormal growth, is convinced that physical factors act at critical phases to produce abnormalities. Areas of high embryonal differentiation and activity would be most vulnerable. Apart from familial and hereditary predisposition,[41] rubella,[30] uterine bleeding,[58] and tubal pregnancy,[10] foetal abnormalities have been produced experimentally by avitaminosis,[2] hypervitaminosis,[29] irradiation, hormones,[82] and other substances.[26] The local effect of anoxia on foetal bowel has been admirably studied by Dr. C. N. Barnard and Prof. J. H. Louw,[51(a)] and further studies on the general effect of anoxia are in progress at the Department of Surgery, University of Cape Town. The exact mechanism whereby these abnormalities are produced is at present conjectural, and until further data accumulate from experimental embryology the propounding of new theories serves little purpose except to perpetuate the confusion.

SUMMARY AND CONCLUSIONS

Intrathoracic duplications of the foregut were considered in the past as pathological curiosities. In fact they are not uncommon. Awareness of the condition is a prerequisite to diagnosis. The macroscopical and histological features of 26 cases are described with illustrative case reports.

Duplications in the thorax arise either from the tracheobronchial or the alimentary systems, and the differentiation is usually easy but by no means absolute. These structures are potentially dangerous and should always be removed without delay, for the complication rate in those of oesophageal origin, and especially in children, is extremely high. A spinal deformity should always be looked for in the chest films and special views obtained if any doubt exists. Associated intestinal duplications should be kept in mind, especially if vertebral anomalies are present and vague abdominal cramps or blood in the stools are noticed.

Existing theories on the origin of duplications are critically analysed and arguments are substantiated by personal embryological and clinical observations. All evidence suggests that duplications are produced in the same way as other abnormalities; that is to say, by physical and chemical factors that act at critical phases in certain areas of the developing embryo.

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HEALTH EDUCATION IN SOUTH AFRICA*

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It is historically true that optimal health conditions cannot be achieved by unilateral governmental action alone. The active intelligent cooperation of all members of a community as individuals must be harnessed with national and local government to produce satisfactory results. Hence the education of the public in health matters is essential.

It has been conclusively shown too that, as in other spheres of education, direction and control must be in the hands of experts, namely experienced medical men assisted by trained educationists and administrators.

Health education has three principal objectives, viz.:

1. To keep citizens informed of the regimen they should follow as individuals, throughout their lives, to maintain themselves in as sound a state of physical and mental health as possible.

2. To make everyone aware of the nature of the community services that are necessary in order to ensure in practice the possibility of reaching that objective.

3. From time to time to publish reliable information, in condensed form and in general terms in regard to results of new methods of treatment and the effects of drugs, operative procedures and so forth.

During the last century the efforts of the medical profession in the sphere of education have rightly been principally directed towards the development of medical schools and the training of doctors, nurses, pharmacists, dentists, radiographers and other health personnel. Remarkable progress has been made the world over. The medical faculties of our South African universities have in a comparatively short time reached a very high standard. All modern teaching methods, both theoretical and practical, have been introduced. Moreover, their postgraduate and research work has already achieved considerable international prestige.

The medical profession in South Africa renders an excellent service to the people in the larger centres as well as in the less populous regions. From personal knowledge and information gained in other ways I am satisfied that no country in the world provides a higher standard of medical practice than ourselves.

For these reasons I propose to confine my remarks to the problems connected with the health education of the public. In my view this is a matter of supreme importance. Hitherto noteworthy efforts have been made; and day schools are doing splendid work in teaching elementary hygiene, and Red Cross, St. John's and Noodhulpliga conduct classes in first aid, home nursing, prevention of accidents and the like. But health education must go much further and should be brought to all groups of society, such as industrial workers, mothers of young children, the aging, and the general population.

Health Education of Natives

In South Africa the situation is complex because of the vastly different backgrounds of our various racial groups. Our Native population needs special consideration. As a group they believe in the power of witchcraft and magic. Patients who under certain circumstances willingly go to European doctors will sometimes remark, 'It is no use taking the patient to a White doctor; this is a disease Whites do not understand'. Problems connected with their sexual life, unrequited love, death, accidents, misfortunes, droughts, fire, lightning, quarrels and enmities are commonly imputed to witchcraft, sorcery or mysticism.

Medicines are used to cause these disasters and others to render them innocuous. The *inyanga* makes the newborn babe witchcraft-proof. Medicines are used to bring the diffident lover to heel. The skin and the hair of the white goat can work wonders. The ritual murder trials recently conducted in the Basutoland courts testify to the profound influence these beliefs still exercise over the Native mind.

Obviously a vigorous campaign of health education designed to replace these pagan ideas by modern health concepts is essential and urgent. The cumulative effect of the personal experiences of hundreds of thousands of Natives every year in hospitals, clinics and doctors' surgeries must certainly be helping. Suitable health films with short captions in the appropriate Native languages impress audiences more than talks. Coloured stills are best of all. Photographs of particular patients on admission to hospital and again on discharge should be taken and exhibited to the Native public. Native commentators, under detailed medical supervision, are the most effective health propagandists. Comparative figures showing for example the improving tuberculosis position should be widely publicized. Moreover, other sections of the population would benefit from health education for reasons quite unconnected with fighting witchcraft.

The film industry has reached a high degree of technical and artistic efficiency. Included in many films are scenes depicting doctors carrying out their daily tasks, but informed members of the audience, viewing such pictures, frequently sense that the atmosphere of the hospital or consulting room is missed. This could be remedied by a deeper appreciation by the actors concerned of the mental approach of an experienced doctor to disease and to his patient. Here then is an important field for appropriate health education.

Instruction of Patients

Many patients of the hospital class undergo major operations not knowing the name of the surgeon or that of the anaesthetist. They are vague about the type of operation performed. They know that their children have had injections, but rarely are they able to state the nature of the said injections.

Patients on discharge from hospital or after attendance at clinics should be given a suitable form duly completed setting out the nature of their illness and the treatment given. In certain circumstances it might be wise and humane to give the information to the relatives and not the patient. At all events this procedure would be a valuable method of health education, enabling the patient to take a more intelligent interest in his own particular trouble. Furthermore, it would be of great assistance to his doctor in the future. These completed forms would become the bases for discussion in family circles and would encourage those concerned to seek perhaps more detailed information.

I was engaged for many years *inter alia* in the medical examination of European personnel for employment. A questionnaire had to be filled in by the candidate. It was evident from the answers given that many regarded tonsillectomy as scarcely worthy of mention in a medical history. This aroused my interest, and I made numerous enquiries. My conclusion was that doctors in their desire to allay the apprehension of the patient and his relatives often give the impression that the operation is a minor procedure, without meaning to do so. Pre-operative instructions and precautions during convalescence would be more faithfully carried out by parents, if the operation were not considered relatively trivial.

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September-October 1949.

There are many wonderful things in the modern world, but none more so than a surgical operation. We doctors sometimes become so used to the triumphs of surgery that we cease to marvel.

Productivity of Health Expenditure

Expenditure on health projects, hospitals, chemical and bacteriological laboratories and clinics is in financial circles often referred to as 'unproductive'. This is a very unfortunate adjective used in this connection, as it leads the less discerning into thinking that 'unproductive expenditure' is 'unnecessary expenditure'.

Modern industrialization followed the advance of medical science. It did not precede it. Vast areas now producing great wealth were practically uninhabitable until malaria and yellow fever were brought under control. Because of these diseases it was necessary to suspend the construction of the Panama Canal until this control had been achieved. The mining industries of the world would collapse without efficient medical organization. It is interesting to note that excellent provision was made for hospitalization and the safeguarding of the public health in the Free State gold fields before mining operations started.

So there are great and powerful industries that act upon the view that sound medical services for its personnel are part and parcel of industry as a whole. There are, alas, many instances where this enlightened policy has not yet been adopted. Health education should therefore be geared to influence leaders of industry, commerce and civic affairs as well. Industrial medicine is now an important part of the work of our profession and up-to-date information would be of great value to industry.

Health Education Policy

A more vigorous health education policy would help the finances of the State by decreasing the demand for hospital beds. Domiciliary treatment could be extended if the public were better informed.

Great benefit, on a long term basis, would accrue to the nation, if every school child from Standard VI onwards were given a short course in the history of medicine. This is an intensely interesting subject and would create the right attitude of mind in our future citizens towards their own health and that of their community.

The Doctor

As a profession we are engaged in the alleviation of human suffering and the prolongation of life. I submit we have carried out these duties with outstanding success—so much so that we are even accused of creating population problems.

Most enlightened people realize that health is their greatest economic asset, but we doctors have not as yet convinced enough of our fellow citizens that what we stand for is sound business and makes for economic stability. We have the facts to prove such a contention and the time is now ripe for us to do so.

Many instances come to the notice of the public which indicate a lack of an effective system of health education. A witness at a recent inquest, according to press reports, said that 'he did not call medical help because the Bible forbade him to do so'. Deaths from diphtheria of children who have never been immunized are still reported.

A contributor to a well-known daily newspaper, when discussing the side-effects of some recently discovered drugs, said 'Yet the best and most humanly helpful of doctors remain extraordinary cagey about this'. Healing cults of all kinds make extravagant claims and often mislead the public. These statements call for reply and clarification from an authoritative source. It is a pity that the medical point of view is not expressed for the guidance of the public. A great deal of credence is given to the cults and it is regrettable that their claims should be ignored and not refuted.

The best form of health education is that given by the general medical practitioner to his patients, but his task would be made easier if his patients through systematic health education were better informed before they become patients. Most doctors are very busy people and they lead exacting and strenuous lives in the practice of their profession, and carry grave personal responsibilities. Indeed I am satisfied that no member of the community works harder than a busy medical practitioner. Even when he is off duty he has invariably some case under observation which makes complete mental relaxation difficult. So much is this the case that many doctors are actually through force of circumstances debarred from carrying out in their own lives what they recommend for their patients. There are very few doctors who do in practice enjoy regularity of meals, rest or exercise. Ask any doctor's wife and you will be given the true picture.

It would seem, therefore, that just as the medical education of students is in the hands of a relatively few members of our profession, so from a practical point of view systematic education of the public could most efficiently be carried out by a group of our profession interested in such matters.

The Medical Association

In February 1959 through the courtesy of the Southern Transvaal Branch of the Medical Association of South Africa, the lay press published in story and in pictures an account of a dramatic and delicate heart operation performed in Johannesburg. The operation was indeed a great and splendid achievement. From a health-education angle the publication of the details was a great success, giving prestige to the profession and demonstrating an advance of significance in medical science.

Health education of the public must be handled with wisdom and knowledge; an injudicious reply to a question might upset the doctor-patient relationship. Hence it is important that health education should be under the direction of medical personnel. What the public needs more than anything else is a clearer notion of the generally accepted principles of medicine. The general practitioner is there to deal with the complaints of the individual patient.

It is not the purpose of health education to give medical advice gratis but rather, through information, demonstration and illustration, to create, a confidence in the public mind in scientific medicine. It is submitted that great blessings will flow from a more vigorous and systematic plan of health education. The related problems of medical ethics and advertising could be controlled and regulated satisfactorily.

The attitude of the Southern Transvaal Branch towards the making public of details of the heart operation mentioned above encourages me to suggest that the Federal Council of the Medical Association of South Africa should appoint a committee to consider the existing position in this country of health education and the making public of medical matters, and to discuss ways and means of making authoritative medical opinion more readily and more generally available to the public than is the case at present. I feel certain that the Government and the public would welcome such statements.

ASSOCIATION NEWS : VERENIGINGSNUUS

APPOINTMENT OF ASSISTANT EDITOR

At the recent Meeting of Federal Council in Pretoria, the Head Office and Journal Committee recommended that Dr. R. L. Kleinman, of Cape Town, be appointed to the post of Assistant Editor of the *Journal*. Federal Council accepted this recommendation *nem. con.*

Dr. Kleinman has had some considerable experience of journalistic work. While at the University of Cape Town, where he obtained the degree of M.B., Ch.B. with distinction in the first professional examination, he was a member of the Editorial Board of the University magazine '*Varsity*. During his student years Dr. Kleinman was University correspondent for several newspapers, and he has continued his connection with these papers by doing occasional reporting and writing leading articles on medical and other subjects. Since June 1959 he has been Editor of the Newsletter of the Cape Western Branch.

Dr. Kleinman has been a member of the Medical Association since he graduated and has been a member of the Contract Practice Committee of the Cape Western Branch, a member of the *ad hoc* Press Liaison Committee, and is a member of the Branch Council.

AWARDS

Federal Council has decided to award the Association's Bronze Medal to Drs. C. Adler, E. Meltzer and H. Grant-Whyte. Emeritus membership of the Association was conferred on Dr. J. Pratt-Johnson, Prof. I. W. Brebner and Dr. B. Weinbren.

DINNER

A very pleasant reception and dinner at the Pretoria Club was arranged for members attending the Federal Council Meeting by the Chairman of Federal Council, Dr. J. H. Struthers, and the President of the Association, Dr. P. F. H. Wagner. An invitation was extended to the Secretary for Health, Dr. J. J. du Pré le Roux, who is to retire shortly, to be present as a guest of honour. Invitations were also extended to Drs. Orenstein, Harvey Pirie and Sichel, who had all held the combined posts of Chairman of Council and President of the Association at one time. Drs. Orenstein and Sichel were present but, owing to indisposition, it was not possible for Dr. Harvey Pirie to be present.

MEDICAL RESEARCH COUNCIL

NATHAN TRUST RESEARCH FELLOWSHIP

The Medical Research Council of Great Britain invite applications for a Research Fellowship provided through the generosity of the Nathan Trust. This appointment is being made under the auspices of a special committee set up by the Council for the evaluation of different forms of therapy in bone sarcoma. Candidates should be clinically experienced British medical graduates, who should preferably have some knowledge of research methods in clinical medicine, surgery, pathology or radiology; and should be willing to engage in this investigation for at least 2, or possibly 3 years, if the progress of the research should so require.

The Fellowship is tenable in the United Kingdom and will provide a salary of £1,500 to £2,000 *per annum* according to age and experience, with superannuation provision in addition; the appointment will be made for 1 year in the first instance, but is renewable for up to 3 years subject to satisfactory progress. Applications, giving a full *curriculum vitae*, a list of publications, and the names of 2 referees (2 signed testimonials in the case of candidates overseas) should be sent to the Secretary, Medical Research Council, 38 Old Queen Street, Westminster, London, S.W. 1, not later than 30 April 1960.

PASSING EVENTS : IN DIE VERBYGAAN

Pensions for Self-employed Persons. Members will have noted with interest the reference made by the Minister of Finance in his Budget Speech to the allowance of £300 *per annum*, which is available for pension purposes, to self-employed persons. Many pension schemes may be brought to the notice of practitioners in the immediate future and members are reminded that the Association is going into the question of a suitable pension scheme applicable to persons in private practice. In due course it is hoped that an announcement will be made in the *Journal*.

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Dr. Stuart J. Saunders, M.B., Ch.B. (Cape Town), M.R.C.P. (Lond.), F.C.P. (S.A.), has commenced practice as a physician at Medical Centre, Heerengracht, Cape Town. Telephones: Rooms 2-7656, residence 69-2924. Dr. Saunders was formerly Senior Medical Registrar at Groote Schuur Hospital and during 1959 he held appointments at the Postgraduate Medical School of London at Hammersmith Hospital, where he paid special attention to diseases of the liver and kidney. Dr. Saunders was also the Cecil John Adams Travelling Fellow in Medicine for 1959.

Dr. Stuart J. Saunders, M.B., Ch.B. (Kaapstad), M.R.C.P. (Lond.), L.K.I. (S.A.), het as internis begin praktiseer te Mediese Sentrum, Heerengracht, Kaapstad. Telefone: Spreekkamer 2-7656, woning 69-2924. Dr. Saunders was voorheen Senior Mediese Registrateur aan die Groote Schuur-Hospitaal en gedurende 1959 het hy betrekkings beklee aan die Nagraadse Mediese Skool van Londen te Hammersmith-Hospitaal, waar hy veral aandag gegee het aan siektes van die lewer en die niere. Dr. Saunders was ook die Cecil John Adams Reis-Genoot in die Medisyne vir die jaar 1959.

* * *

Home Swimming Pools can be Dangerous. Home swimming pools and plastic playpools which are rapidly increasing in popularity hold an element of danger, according to a recent article in a publication of the American Medical Association, *Today's Health*. Written by an instructor at the New York University, Beatrice Schapper, it says that with large numbers of children and adults swimming in their own or in neighbour's back yards, more people than ever face the possibility of accident. Apart from drownings, doctors attribute many colds, ear and nose infections, skin troubles, and other diseases to home swimming pools, where the basic principles of water sanitation are not observed.

The article offers the following suggestions: Keep the swimming water as pure as tap water. Always use a filter. Baths should be backwashed and flushed out every week. Add some form of chlorine to the water. Even with fresh water every day, diseases spread without chlorine, says the article. Water in splash pools and the smaller portables should be changed daily.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.) Joint Lectures. The next lecture in this series will be held on Wednesday 30 March at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Prof. I. Boerema, Professor of Surgery, University of Amsterdam, The Netherlands, will speak on 'Hiatus hernia'. All members of Medical Association are welcome.

* * *

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 4 April at 5.10 p.m. in the Institute Lecture Theatre. Dr. H. S. Gear, formerly Assistant Director General of the World Health Organization will speak on 'The research policies of the World Health Organization'.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Wednesday 30 March at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. The general subject of the meeting will be 'The relationship of cigarette smoking and ischaemic (coronary) heart disease'. Dr. B. Bronte-Stewart will discuss 'General factors', Dr. L. Krut 'Taste thresholds' and Mrs. M. Perrin 'Food preferences'. All who are interested are invited to attend this meeting.

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Mr. J. Roger Boulle, M.B., Ch.B., F.R.C.S., F.R.C.S.E., has commenced practice as a specialist surgeon, in partnership with Mr. Gerald Kane, at 612 Sanlam Buildings, Smith Street, Durban. Telephones: Rooms 25860, 29183; residence 31459; emergency 63737. For the past 2 years Mr. Boulle was a registrar at King Edward VIII Hospital, Durban, and had earlier spent some 9 years doing postgraduate work at various centres in the UK.

* * *

Southern African Cardiac Society. A meeting of the Cape Province Branch was held at Groote Schuur Hospital on 4 February 1960. Dr. W. Beck gave a talk on dye studies and their application to the diagnosis and investigation of heart disease. Dr. C. Barnard spoke on surgery of the mitral valve and illustrated his address with films.

The Chairman, Dr. M. Nellen, announced that it is hoped to hold a South African Cardiac Congress in Johannesburg on 11 and 12 July 1960. Further details may be obtained from Dr. L. Braudo, 904 Medical Centre, Jeppe Street, Johannesburg. The French airline, UAT, has offered to bring out a French cardiac surgeon or cardiologist if this Congress is held.

It was announced that a South African Society of Cardiac Technicians has been formed, with Mr. L. W. Piller, of Groote Schuur Hospital, as National Chairman.

A tie has been designed for the International Cardiac Society at their request, and the meeting approved the design.

* * *

Postgraduate Course for General Practitioners. The University of the Witwatersrand Faculty of Medicine and Medical Graduates Association have organized a full-time intensive postgraduate

course for general practitioners from Monday 18 July to Saturday 23 July 1960. The course will consist of practical demonstrations, ward rounds and symposia. Senior members of the Hospital and University Departments will participate. The course will include paediatrics, anaesthetics, dermatology and orthopaedics, in addition to general medicine, surgery, and obstetrics and gynaecology.

The fee for the course is £5 0s. 0d., payable in advance with the application. Assistance will be given in arranging accommodation for those desiring it. Only a limited number of practitioners can be accepted. Applications should be made to the Medical Graduates Association, Medical School, Hospital Street, Johannesburg, not later than 16 April 1960.

* * *

South African Paediatric Association (M.A.S.A.), Cape Town Sub-Group. The next meeting of this Sub-Group will be held on Tuesday 12 April in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape, at 8.15 p.m. Dr. R. Hoffenberg will speak on 'Recent trends in endocrinology'. Visitors will be welcome.

The Annual General Meeting of the Sub-Group will also be held during this evening.

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Advertisements for Contract Appointments. The attention of members of the Association is drawn to the possibility of advertisements for contract appointments appearing in journals other than the *South African Medical Journal*. When an advertisement for an unapproved appointment is published in the *South African Medical Journal* a notice is placed at the same time advising practitioners to communicate first with the Honorary Secretary of the Branch concerned before applying for the appointment. When advertisements are published in other journals, there is no indication of the acceptability or otherwise of the appointments advertised. It would therefore be in the interests of practitioners for them to ascertain from the local Branch of the Association whether an appointment advertised in another journal carries the approval of the Association or not.

* * *

Dr. P. Packer, M.Med. (Otol.) (Cape Town), will join Dr. F. F. Petersen in specialist practice at 914 Medical Centre, Heerengracht, Cape Town, on 1 April 1960. Telephones: Rooms 3-0018, residence 77-9557. Dr. Packer was formerly Registrar in the Ear, Nose and Throat Department, Groote Schuur Hospital, Cape Town, and has recently returned from a postgraduate study tour of Germany and the UK.

Dr. P. Packer, M.Med. (Otol.) (Kaapstad), sal by dr. F. F. Petersen aansluit in 'n spesialis-praktyk te Mediese Sentrum 914, Heerengracht, Kaapstad, op 1 April 1960. Telefone: Spreekkamer 3-0018, woning 77-9557. Dr. Packer was voorheen Registrateur in die Oor-, Neus- en Keel-departement, Groote Schuur-Hospitaal, Kaapstad, en hy het onlangs teruggekeer van 'n nagraadse studiereis in Duitsland en die V.K.

WEEK-END COURSE IN PSYCHIATRY FOR GENERAL PRACTITIONERS

It has been decided to invite general practitioners to attend a week-end course in 'Psychiatry for the general practitioner', at Tara Hospital from 20 to 22 May 1960; the course is being held in collaboration with the Medical Graduates' Association of the University of the Witwatersrand. The registration fee will be £3 3s. 0d., and the proceeds will go to the Tara Hospital Library.

PROGRAMME

Friday 20 *May*

6.00 p.m. Reception and sherry.
7.00 p.m. Dinner.
8.00 p.m. Opening address: Prof. L. A. Hurst 'Psychiatry in general practice' and Dr. H. Moross 'The place of psychiatry in health services.'

Sunday 21 *May*

9.00 a.m. Physical methods of treatment. Dr. M. Feldman.
10.15 a.m. Tea.
10.30 a.m. Psychiatric emergencies in general practice. Dr. A. Sidley.
12.00 p.m. Case presentation. Drs. D. Perk, M. Feldman, B. W. Levinson, and B. Braude.
1.00 p.m. Lunch.
2.00 p.m. Psychiatric treatment (*a*) Psychopharmacotherapy. Prof. L. A. Hurst. (*b*) Presentation on psychotherapy. Drs. M. Klass and L. S. Gillis.
3.30 p.m. Tea.
4.00 p.m. Anxiety in children. Dr. F. Reinhold.

Sunday 22 *May*

9.00 a.m. Common psychosomatic conditions. Prof. G. A. Elliott.
10.15 a.m. Tea.
10.30 a.m. Psychological problems in marriage. Dr. F. Frankel.
12.00 p.m. Film on alcoholism followed by discussion. Drs. H. E. van Hoepen and M. C. Frame.
1.00 p.m. Lunch.
2.00 p.m. Psychiatric problems in the aged. Dr. D. Perk.

Applications to attend the course should be made to the Medical Superintendent, Tara Hospital, P.O. Box 13, Saxonwold, Johannesburg, as soon as possible.

WORLD LIST OF INTERNATIONAL MEETINGS

Following is a list of international medical meetings to be held during the period July - December 1960. Certain additional information to that published in the *Journal* of 7 November 1959 (**33,** 953) is given for meetings taking place before July 1960. Alterations and additions to this list will be published as soon as they become available.

Fourth Congress of the International Society for Skiing Injuries, Garmisch-Partenkirchen, Germany, 9 - 11 April 1960. Paul Konig, Secretary-General, Orthopädische Poliklinik der Universität München, Pettenkoferstrasse 8a, Munich, Germany. (With an International Competition on Mountain Life-saving.)

Symposium on Microbiological Genetics, London, 12 - 15 April 1960. Prof. B. W. Lacey, Department of Bacteriology, Westminister Medical School, London S.W. 1, England.

Third International Congress on Medical Records, Edinburgh, 24 - 30 April 1960. A. E. J. Turner, Association of Medical Records Officers, United Bristol Hospitals, Royal Infirmary Branch, Bristol 2, England.

International Health Congress, 67th, Torquay, England, 25 - 29 April 1960. Royal Society for the Promotion of Health, 90 Buckingham Palace Road, London S.W. 1, England.

International Congress of Military Medicine and Pharmacy, 16th, Teheran, 25 April - 1 May 1960. Service de Santé de l'Armée, Ministère de la Défense Nationale, Teheran, Iran. With the 22nd Session of the International Office of Documentation of Military Medicine.

Fifth International Congress of Legal Medicine and of Social Medicine, Vienna, April 1960. Prof. William Holczabeck, Secretary General, Sensegasse 2, Vienna, Austria.

International Cancer Cytology Conference, Mexico, D.F., 2 - 11 May 1960. Dr. Robert Nesbitt, Secretary, US Office, International Conference, Union University, Albany, N.Y. Meets with the 10th Inter-American Congress of the Pan-American Medical Association.

Psychotherapy Week, 10th, Lindau, Germany, 6 - 10 May 1960. Secretariat, Dienerstrasse 17, Munich 2, Germany.

Aerospace Medical Association, 31st Annual Meeting, Miami, 9 - 11 May 1960. Dr. William J. Kennard, Secretary-Treasurer, 96 Washington National Airport, Washington D.C.

International Planned Parenthood Federation, 2nd Regional Conference for Europe, Near East and Africa, Netherlands, 11 - 17 May 1960. 69 Eccleston Square, London S.W. 1, England.

International Association for Bronchology, 10th Congress, Lyon, 20 - 21 May 1960. Prof. Dr. Pierre Galy, Secretary General, 16 rue Emile-Zola, Lyons, France.

International Congress of Surgeons, Israel section, Post-Congress International Conference of Surgeons, Tel-Aviv and Jerusalem, 21 - 23 May 1960. Dr. Max Thorek, Secretary-General, International College of Surgeons, 1516 Lake Shore Drive, Chicago 10, Ill., USA. Follows the 12th Biennial Congress, Rome, 15 - 18 May.

International Medical Society of Endoscopic Photocinematography, Television and Radiocinematography, International Symposium, London, 24 - 26 May 1960. Brian Stanford, 54 Upper Montagu Street, London W. 1, England (by invitation).

World Health Organization, Meeting on Tuberculosis, Sydney, May 1960. Palais des Nations, Geneva, Switzerland.

Seminar on the Employment of the Physically Disabled, Dubrovnik, Yugoslavia, May 1960. World Veterans Federation 16 rue de Hamelin, Paris 16e, France.

Pan-American Medical Women's Alliance, 7th Congress, San Juan, Puerto Rico, 3 - 8 June 1960. Dr. Eva Dodge, Corresponding Secretary, 2124 W. 111th Street, Little Rock, Ark., USA.

International Conference on Live Poliomyelitis Vaccines, Washington, D.C., 6 - 10 June 1960. Congress Secretariat, c/o Pan American Union, 1501 New Hampshire Avenue N.W., Washington 6, D.C., USA.

Society of Biological Psychiatry, 15th Annual Convention, Miami Beach, Fla., 10 - 12 June 1960. Dr. G. N. Thompson, 2018 Wilshire Blvd, Los Angeles 57, Calif., USA.

American Medical Association, Annual Meeting, Miami Beach Fla., 13 - 17 June 1960. Dr. F. J. L. Blasingame, Executive Vice-President, 535 N. Dearborn Street, Chicago 10, Ill., USA.

Fourth International Congress of Clinical Pathology, Madrid, 13 - 17 June 1960. Dr. J. Aparicio Garrido, Secretary General, Facultad de Medicina, Pabellon Nr. 2, Cuidad Universitaria, Madrid, Spain.

Food and Agriculture Organization of the United Nations, Economic Commission for Europe, Study Group on Vocational Training and Prevention of Accidents, Geneva, 13 - 17 June 1960. International Agency Liaison Branch, Office of the Director General, Viale delle Terme di Caracalla, Rome, Italy.

International Academy of Pathology, Scientific Meeting, London, 20 - 24 June 1960. Dr. F. K. Mostofi, Armed Forces Institute for Pathology, Washington 25, D.C., USA.

Ciba Foundation Symposium on Sleep, London, 27 - 29 June 1960. (By invitation.) Ciba Foundation, 41 Portland Place, London W. 1, England.

International Society of Geographical Pathology, 7th Conference, London, 28 - 30 June 1960. Prof. J. S. Young, Secretary, British National Committee, ISGP, c/o Department of Pathology, Forresterhill, Aberdeen, Scotland.

Association of Physicians of East Africa, Annual Conference, Nairobi, June 1960. J. Michael Vaizey, M.D. (Cantab.), Hon. Secretary, of the Association, P.O. Box 43, Kampala, Uganda.

United Nations Educational, Scientific and Cultural Organization. Meeting of consultants to advise UNESCO on its Cell Biology Programme, Louvaine, Belgium, June 1960. Place de Fontenoy, Paris 7e, France. In connection with UNESCO's Symposium on Cell Biology and Growth Inhibition.

Fourth International Conference on Goitre, London, 5 - 9 July 1960. Dr. Selwyn Taylor, 3 Roedean Crescent, Roehampton, London S.W. 3, England.

Twelfth Pan-American Tuberculosis Congress, Bahia, Brazil, 10 - 14 July 1960. Prof. Fernando D. Gómez, 26 de Marzo 1065, Montevideo, Uruguay.

International Congress on Congenital Malformations, 1st, London, 18 - 22 July 1960. Dr. W. Bradley, Secretary General, 67 New Bond Street, London, W.1.

International Poliomyelitis Conference, London, 18 - 22 July 1960.

First International Congress of Endocrinology, Copenhagen, 18 - 23 July 1960. Dr. S. G. Johnsen, Secretary General, Hormone Department, Statens Seruminstitut, Copenhagen S, Denmark.

Royal Society, Tercentenary Celebrations, London, 18 - 26 July 1960. Dr. D. C. Martin, Secretary, Tercentenary Celebrations Committee, Burlington House, London, W. 1.

Third International Conference on Medical Electronics, London, 21 - 27 July 1960. Institution of Electrical Engineers, Savoy Place, London, W.C. 2.

International Conference on Mental Deficiency, London, 24 - 31 July 1960. Harvey A. Stevens, 301 Troy Dr., Madison 4, Wisconsin, USA.

London Conference on Scientific Aspects of Mental Deficiency, London, 24 - 29 July 1960. Harvey A. Stevens, P.O. Box 3128, Madison 4, Wisconsin, USA.

Thirteenth International Congress on Occupational Health, New York, 25 - 29 July 1960. James W. Muckell, 527 Madison Ave., New York 22, N.Y.

Ninth International Congress of Catholic Physicians, Munich, 25 July - 1 August 1960. Dr. Pius Müller, Herzog-Max-Strasse 13, Bamberg, Germany.

International Congress on Alcohol and Alcoholism, 26th, Stockholm, 31 July - 5 August 1960. Archer Tongue, Secretary General, Bureau International contre l'Alcoolisme, Case Gare 49, Lausanne, Switzerland.

International Congress of Psychology, 16th, Cologne, 31 July - 6 August 1960. Prof. Otto. Klineberg, Columbia University, New York 27, N.Y., USA.

Sixth International Congress of Anthropological and Ethnological Sciences, Paris, 31 July - 7 August 1960. Prof. Henri Vallois, Directeur, Musée de l'Homme, Palais de Chaillot, Pl. du Trocadéro, Paris 16e, France.

Association for Research into Periodontal Diseases, 16th International Congress, Vienna, July 1960. Prof. S. Loos, Kolingasse 4, Vienna 9, Austria.

Congress of French Speaking Psychiatrists and Neurologists, 58th, early July 1960. Dr. Paul Cossa, Secretary General, Congrés de Psychiatrie et de Neurologie de Langue Française, 29 boulevard Victor-Hugo, Nice, France.

Second International Congress of Neuro-psychopharmacology, Basel, July 1960. Dr. H. C. B. Denber, 1750 Grand Concourse, New York 57, N.Y., USA.

(To be continued)

CORRESPONDENCE : BRIEWERUBRIEK

CLINICAL HYPNOSIS

To the Editor: I should be grateful if you would publish the following case histories which I consider of interest.

The medical profession and the general public as a whole appear to be under many misapprehensions regarding medical hypnosis. This is due to extravagant claims which have been made for hypnosis. Hypnosis definitely has a part to play in medical treatment, but how big that part is depends largely on the technique used and on the selection of suitable cases.

The commonly described technique of applying hypnosis for the removal of symptoms by prestige suggestions, where the operator sets himself up as omnipotent, so frequently fails that ridicule is poured on hypnosis. However, if we apply hypnosis to facilitate psychotherapy, we find that we have a powerful and rapid therapeutic aid.

The cases to be described illustrate: Regression to an earlier age, revivifiction of experiences, and phantasy and dream induction. No claims are made for permanent cures, but the cases are described to show how patients can be helped by hypnosis, keeping in mind that there are very few illnesses of organic or nervous origin which can be permanently cured so as to preclude recurrence.

Case 1

A girl aged 18 complained of severe stuttering since babyhood. Various forms of speech therapy had been tried at school with no success. At the first session hypnosis was induced to the stage where analgesia could be demonstrated. At the second session the trance was deepened sufficiently for her to open her eyes and read from a book without breaking the trance. Suggestions were given during this trance that she would be able to read without stammering while under hypnosis. She read perfectly for several minutes.

At the third session hypnosis was induced to a sufficiently deep stage for her to be able to regress to an earlier age. We do not know whether the regression that takes place under hypnosis is true or only apparent, but it serves a very useful purpose when old repressed subconscious material is being sought. Regression took place in stages, a few years at a time, and at each age she was asked to say a few words. She continued stammering until she was regressed to the age of 2 years. The regression was then moved to the stage of 18 months, and she spoke very childishly, but without a stammer. From 18 months she was advanced a month at a time, and the change from normal speech to disabled speech occured at 23 months.

At the next session it was explained to her that something must have happened to her at 23 months. She was taught the technique of phantasy and dream production. She was then regressed to the age of 23 months and asked to have a dream that would recapture the occurrence which coincided with her first attack of speech impediment. She then described a scene where she was playing with her brother who is a few years older than she is; he was stuttering and she was copying him.

Before being awakened she was told that now that she knew what the cause of her stuttering was she would no longer stutter herself. She would become confident in her ability to control her speech, in conversation as well as reading. Further suggestions of confidence were given after she was awakened.

She was seen 6 weeks after the last session and was still speaking fluently.

Case 2

A female aged 43, the mother of two children, aged 21 and 15 years. The history of her case, as given originally, is as follows:

For the past 3½ years she has had persistent headaches, day and night, mainly on the left side of her forehead involving deep pain in the left eye. The pain, which has been almost continuous, showed a partial and temporary response to analgesics and ergotamine preparations. There have been acute exacerbations during which time she had severe attacks of vomiting. She has been incapable of carrying out her household duties for the past 3 years. She has lost her appetite almost completely, and 65 lb. in weight. When first seen she weighed 102 lb., being 5 ft. 4 in. tall. She also had severe insomnia.

She had lost interest in her person and her family. On questioning she admitted to having had headaches since a child at irregular intervals; the headaches were of moderate severity, but not persistent. The beginning of the severe headaches coincided with some domestic trouble with her husband. This, however, was patched up 3 years ago.

She has been attended during the 3½ years by physicians and psychiatrists. A surgeon had seen her with a view to possible thyroidectomy and a neurosurgeon had seen her with a view to possible neurosurgery. Both refused to operate. No organic cause had been found for the symptoms. Her condition was labelled 'migraine'

When first seen the patient presented as an emaciated, shabbily dressed, drowsy woman who appeared to be very depressed and completely uninterested in her surroundings. She spoke very slowly and was only interested in telling of her boring pain in the side of her head and eye.

Light hypnosis was induced easily and carried to a sufficient depth for analgesia of her calf muscles to be demonstrable. At this stage temporary analgesia of her face, head and eyes, was induced and she was rapidly awakened so as to demonstrate the possibility of removing her headache under hypnosis. She agreed that the headache was almost gone. It was explained that this was a temporary relief, and that further relief would be forthcoming if she would cooperate in a treatment schedule.

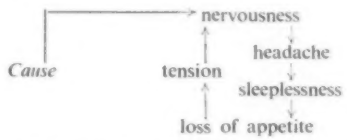
Hypnosis was re-introduced. This time suggestions were given to the effect that she would sleep normally that night without any tablets and in spite of her headache, which would not disturb her sleep. She was then re-awakened.

At the next session the following day she reported that she slept well, but the headache was unabated. She seemed to be more interested in herself and her surroundings. A trance was induced and she was regressed 3½ years, to the time when the headaches began. She was then taught how to develop phantasies and dreams on command. She was instructed to have a happy dream followed by a bad dream. The happy dream would relieve and the bad dream, she was told, would again bring back her headache. She was instructed to tell the dreams without awakening. The happy dream was of dancing girls on a stage, which relieved her headache. The unhappy dream concerned a particular dancer, who was struck down by a woman who ran up onto the stage. This dream caused an agonizing headache to appear. A further happy dream induced was of a man and a lady dancing. Its unhappy equivalent was when a second woman appeared and the first dances were rejected; this also caused an exacerbation of the headache.

While still in the trance she was told to identify the man and the first woman who were dancing. She said they could be herself and her husband.

It was explained to her that the causes of her headaches date back to a quarrel with her husband and that further light would be thrown on the matter at the next session. She was again given suggestions for sleeping well at night.

At the next session she reported that she slept well, but the headaches were still unaffected. The mechanisms of the vicious circle were then explained to her in the waking state. It was pointed out that the vicious circle, once set in motion, continued on its own although the initial cause of the tension had been removed, thus:



It was further explained to her that the quarrel with her husband was only the exciting cause and not the initial cause of the headaches. She had had headaches before the 3½ years of severe headache, and we would together probe into the past and eliminate the original cause.

Under deep hypnosis with regression and dream production, the following came to light: She had her first attack of headache in a classroom with her geography teacher. She then was 11 years old. The teacher had a cane on the desk and threatened to cane the girls. The patient did not know her lesson and developed a severe headache. Another scene of headache concerned a quarrel between her elder brother whom she loved very much, and his wife. She wanted to help her brother, but decided that she could not fight against her sister-in-law who was old enough to be, and acted as if she were, her mother.

It was explained to her under hypnosis that each time she came into conflict with authority, she developed a severe headache due to tension, because she did not do anything in self-defence or self-justification. It was not the situation that caused the headache, but the fact that she neither fought nor withdrew. She allowed her own tension to harm her. In future she would feel stronger in herself. Her determination to defend herself would strengthen and she would find that as she made her own will felt by others, they would think better of her and she would feel better herself. Further encouragement with insight into her condition were given in several further treatment sessions, and the results have been very gratifying. She gained 5½ lb. in weight in 3 weeks. She has slept every night without any drugs since the hypnosis began. She now feels well and has begun to do her household chores. She has taken a new interest in her personal appearance.

At the last session she reported only one slight attack of headache during the past week.

She was instructed to dream an unhappy dream under hypnosis and, while in the trance, reported a recurrence of the headache. Instructions were given for a happy dream, and this in turn removed the headache in seconds.

She will be seen at regular intervals to encourage her to rehabilitate herself to her family and friends and her every-day life, and to re-demonstrate from time to time how her headaches were caused by tension, conflict and unhappy events.

It must be stressed again that it is not the hypnotic state itself which helps, but the therapy that is applied under hypnosis, and that the cases which can be treated successfully have to be selected carefully.

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